A STUDY OF DYSLIPIDAEMIA IN HIV PATIENTS RECEIVING HAART

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ABSTRACT

BACKGROUND

Human Immunodeficiency Virus (HIV) was discovered in 1986 in Chennai (India) amongst female sex workers by Dr. Suniti Solomon. Since then, HIV has spread to all parts of the country from the high-risk group to the antepartum population in many states at an alarming rate. The prevalence of dyslipidaemia and other risk factors for cardiovascular disease is significant in HIV/AIDS patients receiving highly active antiretroviral therapy (HAART), ranging from 20% to 80%. In view of the high prevalence of dyslipidaemia and the increased risk for cardiovascular diseases among patients with HIV/AIDS, this is a matter of concern for public health.

MATERIALS AND METHODS

143 patients who had been receiving HAART for a minimum of two years from Rajiv Gandhi Institute of Medical Sciences, Kadapa, during the period of January 2015 to September 2016 were studied. They were divided into 4 regimens groups 1) TEL (Tenofovir, Efavirenz, Lamivudine) 2) TLAR (Tenofovir, Lamivudine, Atazanavir, Ritonavir) 3) ZLE (Zidovudine, Lamivudine, Efavirenz) 4) ZLN (Zidovudine, Lamivudine, Nevirapine). Detailed history, demographic data, anthropometric measurements, serum lipid profile obtained and analysed.

RESULTS

Out of 143 patients, 90 (62.9%) were males and 53 (37.1%) were females. 68 (47.6%) were in the 30-39 years age group accounted for maximum percentage of groups. Based on BMI only 3 (2.1%) were obese, 24 (16.8%) were of overweight. Waist-Hip ratio was abnormal in 117 (81.8%) and 26 (18.2%) were normal. The mean values for patients on TEL regimen are TC is 195.4 mg%, LDL 122.1 mg%, HDL 34.96 mg%, TG 194.02 mg% and TC/HDL is 5.5714. In patients treated with TLAR regimen the mean values of TC are 172.15 mg%, LDL 99.15 mg %, HDL 36.35 mg%, TG 183.35 mg% and TC/HDL is 4.8. In patients treated with ZLE regimen, TC is 201.64 mg%, LDL 123.27 mg%, HDL 35.68 mg%, TG 212.27 mg% and TC/HDL is 5.6364. In patients treated with ZLN regimen, TC is 162.1 mg%, LDL 91.94 mg%, HDL 35.98 mg%, TG 172.54 mg% and TC/HDL is 4.5192.

CONCLUSION

The study showed an increased prevalence of dyslipidaemia in the 30-39 age groups particularly among males. Waist-to-hip ratio is significantly elevated in the HIV-infected patients on HAART. This showed a significant correlation to waist/ hip ratio and the gender. The lipid analysis showed a significant increase in the total cholesterol and LDL in the regimen groups. There was an insignificant increase in total triglycerides, VLDL and fall in HDL levels in the regimen groups. The abnormalities in lipids were more in patients of the efavirenz based regimen.

KEYWORDS

HAART, Dyslipidaemia, Waist/Hip Ratio.

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BACKGROUND

According to NACO there are 2.1 million people living with Human Immunodeficiency Virus (HIV) infection and Acquired Immunodeficiency Syndrome (AIDS) in India.[1] The global pandemic of HIV infection & AIDS is virtually reported from all countries with a global prevalence of 36.7 million and incidence of 2.5 million new cases every year.[2] Patients with HIV or AIDS frequently have alterations in lipid metabolism due to infection with HIV itself.[3] The introduction of antiretroviral therapy (ART) in the mid-1990s led to substantial improvement in the prognosis of HIV/AIDS patients, with a reduction in morbidity and mortality due to opportunistic diseases and consequent improvement of the patient's quality of life.[4,5] However, there is evidence that ART is associated with lipodystrophy syndrome, a disturbance of lipid metabolism characterized by insulin resistance, dyslipidemia, and fat maldistribution, usually presenting as visceral abdominal obesity and cervical fat pad accumulation (buffalo hump).[6] metabolic bone disease

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(osteopenia and/or osteoporosis), and lactic acidosis.[5] ART-associated dyslipidemia is characterized by elevated serum concentrations of total cholesterol (TC), triglycerides (TG), low density lipoprotein (LDL-c), very low-density lipoprotein (VLDL), and apolipoprotein B (apoB), and low levels of high density lipoprotein (HDL-c), constituting an atherogenic lipid profile.[7] The prevalence of dyslipidemia and other risk factors for cardiovascular disease is significant in HIV/AIDS patients receiving ART, ranging from 20% to 80%. Prevalence rates of lipodystrophy vary widely from 11 to 83 percent in cross-sectional studies.[8] These lipid alterations were first described in patients who used antiretroviral regimens containing protease inhibitors, but were later observed in patients who received regimens consisting of nucleoside reverse- transcriptase inhibitors (NRTI) and non-nucleoside reverse-transcriptase inhibitors (NNRTI) also.[9] Some antiretroviral drugs, such as stavudine (d4T), and protease inhibitors (PIs).[10] increase the blood levels of TC, LDL-c, and TGs with variable effects on levels of HDL-c. Nevirapine (NVP) use is associated with increase in HDL-c, whereas increases in TC and TG are observed with use of efavirenz (EFV), particularly with longer duration of therapy. Therefore, the use of HAART raises concerning metabolic disorders and cardiovascular risk in HIV infected patients who now present an extended life expectancy. In view of the high prevalence of dyslipidemia and the increased risk for cardiovascular diseases among patients with HIV/AIDS, which is a matter of concern for public health, this study is aimed to determine the prevalence of dyslipidemia and characteristics of lipid profiles among people living with HIV infection receiving HAART.

MATERIALS AND METHODS

This cross-sectional study was undertaken at Rajiv Gandhi Institute of Medical Sciences, Kadapa. The study was carried out from January 2015 to September 2016 and 143 patients in the age group of 20- 49 years were included in the study who had been receiving WHO recommended HAART for a minimum of two years. Participants used HAART regimens that included nucleoside reverse transcriptase inhibitors (NRTIs): Lamivudine (3TC), Zidovudine (d4T), Tenofovir (TDF) and non-nucleoside reverse transcriptase inhibitors (NNRTIs): Nevirapine (NVP) or Efavirenz (EFV) and protease inhibitors (PIs): Atazanavir (ATV) and Ritonavir (RTV). Patients who changed their regimens during follow-up were not included. Patients with the following diseases such as Diabetes, hypertension, liver disease, renal failure, coronary artery disease, Nephrotic syndrome, Hypothyroidism, Patients on Antihyperlipidaemic drugs, Thiazides Steroids, Beta blockers. Pregnant women, uncooperative and non-willing patients and patients on HAART for less than 2 years.

Patients included in the study were divided into 4 regimen groups.

1. TEL (Tenofovir, Efavirenz, Lamivudine)
2. TLAR (Tenofovir, Lamivudine, Atazanavir, Ritonavir)
3. ZLE (Zidovudine, Lamivudine, Efavirenz)
4. ZLN (Zidovudine, Lamivudine, Nevirapine)

Demographic data regarding, age, gender, ART exposure and the regimen in the previous two years were collected using questionnaire which also included relevant history of Hypertension, Diabetes mellitus, Ischemic heart disease and Cerebrovascular accident. Anthropometric Measurements Body weight, Height, Hip and Waist circumferences were measured. All measurements were read in centimeters (cm) but the height was converted to meters. BMIs were then calculated as weight in kilograms divided by the height in meter squared.

Sample Preparation and Biochemical Assay- 5 ml of venous blood sample was collected by venipuncture from 12 hours overnight fast. The serum levels of TC, HDL-C, LDL-C, VLDL and TG were measured using AU480 BECKMANS random access fully automated auto analyzer at Biochemistry laboratory, RIMS, Kadapa. TG and total TC were evaluated with enzymatic method and HDL-C and LDL-C were analyzed by enzymatic method when triglycerides >400 mg/dL. Low-density lipoprotein cholesterol (LDL-C) was calculated using Friedewald's formula in individuals with triglycerides <400 mg/dL. To assess cardiovascular risk, Castelli's Index. I was calculated using the ratio: TC/HDL-C; a Castelli Index I >5.1 for men and >4.4 for women were considered indicative of an elevated risk.

RESULTS

The following observations were made in our present study.

Regimen/Group	No. of Cases	% of Total Cases
TEL	49	34.3
TLAR	20	14
ZLE	22	15.4
ZLN	52	36.4
Total	**143**	**100**

Table 1. ART Regimen wise Distribution of Cases

Group	No. of Cases	Percentage
20 -29 years	22	15.4
30 -39 years	68	47.6
40- 49 years	53	37
Total	**143**	**100**

Table 2. Age wise Distribution of the Cases

Gender	No. of Cases	Percentage
Male	90	62.9
Female	53	37.1
Total	**143**	**100**

Table 3. Gender Distribution of the Cases

BMI	No. of Cases	Percentage
Under Weight	31	21.7
Normal	85	59.4
Over Weight	24	16.8
Obesity	3	2.1
Total	**143**	**100**

Table 4. BMI among the Cases

Group/Regimen	No. of Cases	Mean CD4 Count	Std. Deviation	Std. Error	F-Sig. Value	Result
TEL	49	235.94	115.375	16.482	0.0001	Significant
TLAR	20	218.8	119.548	26.732		
ZLE	22	169.41	96.472	20.568		
ZLN	52	299.21	96.417	13.371		
Total	**143**	**246.31**	**114.943**	**9.612**		

Table 5. CD 4 Cell Count Among the Study Groups

WC-HC Ratio	No. of Cases	Frequency
Normal	26	18.2
Abnormal	117	81.8
Total	**143**	**100**

Table 6. Waist-Hip Circumference Ratio

	Gender Group	N	Mean	Std. Deviation	Std. Error Mean	t-Sig. Value	Result
W.C/H.C	Male	90	0.9578	0.07604	0.00801	0.05	Significant
	Female	53	0.9204	0.07385	0.01014		

Table 7. Waist-Hip Ratio with Gender Group

Out of 143 patients, 90 (62.9%) were males and 53 (37.1%) were females. 68 (47.6%) were in the 30-39 years age group accounted for maximum percentage of groups. Based on BMI only 3 (2.1%) were obese, 24 (16.8%) were of overweight. Waist-Hip ratio value analysis showed abnormal in 117 (81.8%) and 26 (18.2%) were normal. the mean CD4 count is highest in ZLN regimen (299.21) and least in ZLE regimen (169.41).

Lipid analysis showed a significant increase in total cholesterol and LDL in the regimen groups. There was an insignificant increase in total triglycerides, VLDL and fall in HDL levels in the regimen groups. The abnormalities in lipids were more in the regimen group especially efavirenz based regimen.

		N	Mean	Std. Deviation	Std. Error	F-Significant Value (p- value)	Result
TC	TEL	49	195.41	61.697	8.814	0.001	Significant
	TLAR	20	172.15	41.107	9.192		
	ZLE	22	201.64	39.842	8.494		
	ZLN	52	162.1	41.048	5.692		
	Total	143	181	51.305	4.29		
HDL	TEL	49	34.96	4.765	0.681	0.574	Not Significant
	TLAR	20	36.35	3.345	0.748		
	ZLE	22	35.68	4.145	0.884		
	ZLN	52	35.98	4.518	0.627		
	Total	143	35.64	4.395	0.368		
LDL	TEL	49	122.1	54.52	7.789	0.003	Significant
	TLAR	20	99.15	38.486	8.606		
	ZLE	22	123.27	31.035	6.617		
	ZLN	52	91.94	43.219	5.993		
	Total	143	108.1	47.185	3.946		
TG	TEL	49	194.02	94.618	13.517	0.379	Not Significant
	TLAR	20	183.35	72.565	16.226		
	ZLE	22	212.27	77.756	16.578		
	ZLN	52	172.54	106.474	14.765		
	Total	143	187.52	94.29	7.885		
VLDL	TEL	49	38.57	19.012	2.716	0.375	Not Significant
	TLAR	20	36.65	14.597	3.264		
	ZLE	22	42.32	15.616	3.329		
	ZLN	52	34.23	21.529	2.986		
	Total	143	37.3	19.005	1.589		
tc/hdl	TEL	49	5.5714	1.69558	0.24223	0.001	Significant
	TLAR	20	4.8	1.28145	0.28654		
	ZLE	22	5.6364	1.1358	0.24215		
	ZLN	52	4.5192	1.21252	0.16815		
	Total	143	5.0909	1.47232	0.12312		

Table 8. Lipid Profile in Study Regimen Groups

DISCUSSION

The use of HAART has significantly decreased morbidity and mortality in HIV infected patients leading to an increase in life expectancy. However, the benefits of ART are associated with a wide spectrum of side effects with some clinical manifestations.[11] Lipodystrophy, hyperlipidemias, insulin resistance, hyperglycemia and even overt diabetes has been reported in subjects treated with protease inhibitors (PIs) and nucleoside reverse transcriptase inhibitors (NRTIs).[12] Lipodystrophy characterized by peripheral loss of fat tissue and abnormal fat distribution including the enlargement of dorsocervical fat pad, lipomatosis, breast hypertrophy, and visceral abdominal fat accumulation have recently been reported in HIV-1 patients receiving HAART.[13] Dyslipidemia in the HIV-infected is mostly attributed to HAART treatment. The prolonged surge of pro inflammatory cytokines such as TNF-alpha, IL-1, IL-6 and IFN-alpha observed following the chronic state of HIV infection have been shown to contribute to lipid dysregulation.[14] Cytokines such as TNF- alpha, IL-1, IL-6 and IFN-alpha have also been reported to increase lipogenesis, decrease clearance of circulating LDL and inhibit hepatic lipase activity.[15] Dyslipidemia in HIV-infected patients is due to the changes in lipid metabolism induced by medium to long term exposure to ART. While direct dyslipidemia induced by protease inhibitors (PI) can develop rapidly, other more chronic metabolic changes affecting lipid metabolism can occur with HAART. Shortly after the introduction of HAART, a syndrome of subcutaneous lipoatrophy, central adiposity, dyslipidemia, and insulin resistance, termed HIV-associated lipodystrophy (HIVLD) was noted.[16] This was initially associated with PIs exposure, but subsequently exposure to NRTIs.[17] particularly thymidine analogue NRTIs (NRTIs) such as stavudine and zidovudine were also recognized as being central to the development of this syndrome. Compared to HIV-infected controls without lipodystrophy, individuals with HIVLD tend to have higher total cholesterol, total cholesterol: HDL ratio, LDL-C and triglyceride levels. In this study maximum prevalence in the 30 -39 years age group among HAART regimen groups I-IV (47.6%). Sex distribution in this study showed 90 number of cases are males and 53 number of cases are females and constituted 62.9% of males and 37.1% of females in study regimen groups 1-IV. Study Groups showed values similar to those in HIV Sentinel surveillance 2007 done by NACO[1] and UNAIDS report 2016.[2] Among our study subjects, there was no association between serum lipid levels and gender. However, according to a study from Thailand.[18] on 200 HAART treated patients for an average of 39.35 months, the prevalence of hyperlipidemia was higher in men than in women. The discrepancy with our study may be due to our study participants who were not gender matched between the groups. The patients are divided on the basis of HAART regimen groups and the mean CD4 count is highest in ZLN regimen (299.21) and least in ZLE regimen (169.41). The F-value is significant <0.05 and the difference between the CD4 counts is statistically significant with respect to the regimen groups. In contrary to our study, Dickson Shey et al.[19] in their study found that there was no significant difference observed in the different regimen groups with different CD4+ T cell count categories.

Indeed waist circumference, hip circumference and waist-to-hip ratios which have been shown to be better reflectors of body fat distribution, were significantly elevated in the HIV-infected patients on HAART. Abnormalities in body composition have been reported in 40 to 50 percent of ambulatory HIV-infected patients, according to Lichtenstein KA, Ward DJ et al.[8] The physical appearance of lipodystrophy is more apparent usually by 2 years of HAART.[13] Lipodystrophy is more apparent with the increase of life expectancy, in HIV patients treated with HAART on reaching up to 49.5 years, when treatment is started at the age of 20 years.[20] Anthropometric parameters are important tools for measurement of lipodystrophy including weight, BMI, Waist/Hip ratio and skin fold thickness. Many of the studies failed to show good correlation with lipoatrophy and anthropometry.[21] In this study 21.7% of the cases were underweight and 59.4% had a normal BMI. 16.8% are overweight and 2.1% were obese. Lichtenstein KA, Ward DJ et al.[8] in their study concluded that a decreasing BMI rather than increasing BMI, increased triglycerides, older age and female sex are more prone for lipodystrophy.[21] Waist-Hip ratio value analysis showed normal in 18.2% of the cases and abnormal in 81.8% of the cases. The mean value in males is 0.9578 and in females is 0.9204, which is above the guideline values for men (>0.90) as well as women (>0.80), t-value is significant <0.05, reject null hypothesis and the difference between the W.C/H.C is statistically significant with respect to the gender. This shows Waist-Hip ratio has significant relation to long standing illness and abdominal obesity. Considering the anthropometric measurements observed in this study, Waist -Hip ratio is a better parameter for assessing the visceral adiposity. Low BMI reflects the weight loss with progression of disease and an increasing Waist-Hip ratio denoting the visceral adiposity.

A study done by RA Ngala et al.[22] suggested that the combined use of NNRTI and NRTI resulted in 24% of the subjects experiencing facial fat depletion. However, participants on zidovudine, lamivudine and efavirenz combination therapy had no changes in appearance in terms of fat depletion on the limbs as well as fat accumulation in the breast and buttocks. About 29% had truncal obesity after using the various combinations of NRTI and NNRTI. The association between visceral obesity and NRTI therapy has been reported in several studies.[23] E Frontas, Friis Moller et al.[24] in their study (DAD study first phase) found that dyslipidemia occurred in patients among all HAART groups including Protease inhibitor, NNRTI as well as NRTI. Patients on Protease inhibitor especially dual protease inhibitor had more lipid abnormalities. We found that the prevalence of raised TC in the HAART group was high. This prevalence is higher than that reported from two similar studies in Cameroon.[25] and that found in rural

Ugandans.[26] The prevalence of high LDL-c in our study was similar to the prevalence reported from India.[27] However, the mean LDL-c was significantly higher in the obese. The prevalence of raised TG in our study was lower than that reported in India.[27] Several studies have found that stavudine was more involved in the occurrence of lipid derangements as compared to other NRTIs. However, instead of stavudine, our participants were either on tenofovir or zidovudine. We found no difference in lipid profiles (TC, LDL-c and HDL-c) when participants on tenofovir were compared to those on zidovudine. Similar to the findings of Yone and colleagues in Cameroon.[28]

According to a study by RA Ngala, et al.[22] in 2013 stated that total cholesterol, low density lipoproteins and high-density lipoproteins were significantly raised in the HAART-experienced. The raised HDL level as a result of the combined use of NRTI's and NNRTI's in this study may therefore serve to improve reverse cholesterol transport and therefore bringing the lipoproteins to the physiological level and their use would be much recommendable. Suelen Jorge Souza, et al.[29] study stated that ART regimens promoted distinct alterations in the lipid metabolism of the HIV patients. Protease inhibitors, particularly indinavir and lopinavir, were commonly associated with hypercholesterolemia, hypertriglyceridemia, elevated LDL-c, and reduced HDL-c. Fewer lipid alterations were observed with use of the protease inhibitors atazanavir. Some NRTIs (didanosine, stavudine, and zidovudine) more frequently induced lipid alterations, particularly lipoatrophy and hypertriglyceridemia. However, tenofovir-containing NRTI regimens resulted in a better metabolic profile. Patients using NNRTIs developed hypertriglyceridemia and hypercholesterolemia. The NNRTI nevirapine was particularly associated with elevated concentrations of HDL-c. Adewole et al.[30] in 2010 had a cross-sectional study on NNRTI drugs for 12 months and found that nevirapine promoted raised HDL-c and stabilisation of TC and TG. Lipid profiles should be performed at baseline before commencement of antiretroviral therapy and then periodically through treatment follow-up to monitor any rising trends. The association between HAART and adverse lipid profile has been largely described for regimens that include PIs.[31] but this is contrary to our findings. This may be due to the small number of patients treated with PIs in our study. In our study, TC, LDL AND TC/HDL lipid levels are significant. F-significant values are <0.05, reject null hypothesis. The difference among the lipid profiles of TC and LDL in the study group is statistically significant with respect to regimen groups. The HDL, TG and VLDL are not significant. F- significant values are >0.05, no evidence to reject null hypothesis and there is no significant difference among the lipid profiles of HDL, TG and VLDL in the study group and is not statistically significant with respect to regimen groups. Similar to our findings, a cross-sectional study from India.[32] showed significantly higher prevalence of dyslipidemia in the first line treatment groups. In addition to its benefit antiretroviral drugs have been associated with an abnormal fat redistribution syndrome that might raise cholesterol and triglycerides levels. Compared to each other, the independent effect of the use of NVP and EFV based combinations on serum lipid profile level was not seen among our study participants. On the contrary, a 48 week follow up study in Australia.[33] found that, the increase of HDL-C was significantly larger for patients receiving NVP than for patients receiving EFV, while the increase in TC was lower. The increase of non-HDL-C was smaller for patients receiving NVP than for patients receiving EFV, as were the increases of TG and LDL-C. In addition, a study in our country.[32] found that TC level >200 mg/dl was more common among patients who received EFV than among those who received NVP.[19] In our study, the lipid parameters abnormalities were more in the ZLE regimen and least in ZLN regimen. Analysis of the data in this study showed that all the lipid parameters abnormalities were more pronounced in the group which had nevirapine and stavudine compared to efavirenz and zidovudine-based regimens.

The mean total cholesterol was 195.41 mg%, 172.15 mg%, 201.64 mg% and 162.10 mg% in the TEL, TLAR, ZLE and ZLN regimen groups respectively. The p value is significant. Similarly, there was statistically significant increase in LDL and TC/HDL ratio. Leonardo Calza; Roberto Manfredi; et al[34] in their study showed patients on nevirapine arm did well in both Triglyceride arm and Total cholesterol causing a lowering of both parameters by 23% and 29%, while in efavirenz based arm the results were 9% and 11% reduction after switching from PIs based regimens. Similar observations are found in our study. This results clearly shows a more abnormal lipid profiles in efavirenz based regimen than nevirapine containing regimen. Nevirapine is a better drug substitution for PI in patients with multiple risk factors for cardiovascular event. Total Cholesterol to HDL ratio in this study is 5.57 in TEL regimen, 4.8 in TLAR, 5.64 in ZLE regimen and 4.52 in ZLN regimen. In the DAD study.[35] the mean values TC: HDL ratio was 5.3 in HAART treatment Group. This shows the increased Atherosclerotic and cardiovascular risk in this population. A study done by Max Weyler et al.[35] showed that approximately 40% of patients had an elevated Castelli Index I, indicating an increased risk for atherosclerotic cardiovascular disease in this population. This index is considered a simple approach for lipid risk assessment, the high total cholesterol is a marker for atherogenic lipoproteins and low HDL cholesterol correlates with risk factors of metabolic syndrome. Eoin R Feeney and Patrick W.G Mallon et al.[36] stated that dyslipidemia and lipodystrophy will continue to be major issues for many HAART treated patients for years to come. The recent introduction of new medications with more lipid friendly profiles within existing classes such as darunavir (PI) and etravirine (NNRTI).[37] will broaden the options available to clinicians. In addition, entirely new classes of drugs such as integrase inhibitors (raltegravir) and CCR5 inhibitors (maraviroc).[38] should allow more options both for antiretroviral-naïve patients starting therapy and those needing to switch therapies to avoid dyslipidemia.

Furthermore, the development of newer selective boosting agents to replace ritonavir that lack dyslipidemia is continuing. Uses of HAART regimens are significantly associated with atherogenic lipid profiles. Lipid profile and other cardiovascular risk factors should be monitored in patients on ART so that any negative effects of HAART can be optimally managed and recommend the implementation of well controlled cohort studies for the evaluation of long-term effects of HAART treatment on lipid profiles.

CONCLUSION

Significant metabolic and morphological alterations occur in HIV infected patients especially in patients on HAART. There is a statistically significant increase in the total cholesterol and LDL cholesterol. There is statistically insignificant increase in total triglycerides, VLDL and decrease in HDL cholesterol in HIV patients on HAART. The patients on HAART had an elevated Castelli Index I, indicating an increased risk for atherosclerotic cardiovascular disease in this population. Waist-to-hip ratios which are the key indices for assessing body fat distribution were significantly raised implying that HAART could result in lipodystrophy. Dyslipidemia is more in the efavirenz based regimens compared to nevirapine, tenofovir, lamivudine, zidovudine and atazanavir containing regimens. There is need to assess lipid profiles at baseline before initiation and during therapy to monitor any rising trends. New medications with more lipid friendly profiles within existing drugs such as darunavir (PI), and etravirine (NNRTI) and new classes of drugs such as integrase inhibitors (raltegravir) and CCR5 inhibitors (maraviroc) can be used to avoid dyslipidemia. The results also recommend implementation of well-controlled cohort studies for the evaluation of long-term effects of HAART lipid profiles. Use of HAART regimens are significantly associated with atherogenic lipid profiles. Lipid profile and other cardiovascular risk factors should be screened and monitored in patients on HAART every 6 months, switching to lipid friendly drugs. Lipid modifying drugs as well as change in life style may help to nullify the increased cardiovascular risk and mortality in HIV patients on HAART.

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