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Impact of Highly Active Antiretroviral Therapy (HAAT) on Lipid Profiles of HIV Patients

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Abstract

Highly active antiretroviral therapy (HAART) has resulted in a significant reduction in morbidity and death in HIV patients. However, it has also resulted in the appearance of significant short- and long-term negative effects. The complication of HAART that is currently generating the most interest and concern is an elevated cardiovascular risk.The aim of this study was to assess the impact of highly active antiretroviral therapy on fasting blood lipid profiles in HIV patients. This prospective, observational study was conducted in the ART Centre of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh, between January 2020 and June 2021. A total of 49 HIV sero-positive patients (diagnosis based on rapid diagnostic test) attending ART Centre of Bangabandhu Sheikh Mujib Medical University (BSMMU) Hospital were included in this study. We found that 9.7% had TC ≥200. The majority 80.6% patients belonged to HDL-C <40 (mg/dl) and the mean was 31.4±9.26 (gm/dl). Regarding LDL, 9.7% of patients had LDL-C (>130 mg/dl), and the mean was 85.34±28.79 (gm/dl). About half 51.6% of patients belonged to TG >150 (mg/dl) and the mean was 154.46±59.62 (gm/dl). We found that 87.1% of patients developed dyslipidaemia before treatment and 83.9% after HAART. TG levels were significantly elevated after HAART, but TC, HDL-C, and LDL-C levels almost remained the same between baseline and after HAART. Development of dyslipidaemia was found in more than two-thirds (83.9%) of the patients after HAART. HIV-infected patients receiving WHO recommended first-line of HAART have a high prevalence of lipid profile derangement.

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Introduction

A huge number of HIV patients are living throughout the world. It is stated that approximately 37.7 million people across the globe lived with HIV/AIDS in 2018, among them around 27.5 million people were accessing antiretroviral therapy.[1] In the year 2018, about 1600 people were newly infected with HIV, and 3045 were under HAART coverage in our country.[2] In the current world with the advancement of ART patient's life expectancy is increasing day by day due to a magnificent decline in overall mortality from AIDS-related illness in immunodeficiency-related events and death.[3,4] Highly active antiretroviral therapy (HAART) has resulted in a significant reduction in morbidity and death in HIV patients.[5,6] However, it has also resulted in the appearance of significant short- and long-term negative effects.[7,8] The complication of HAART that is currently generating the most interest and concern is an elevated cardiovascular risk.[9,10] The relationship between HAART and the development of dyslipidemia has been extensively researched and proven.[11-13] In HAART era the major challenge is to combat non-AIDS-relateddeaths and morbidity from premature e cardiovascular disease, liver disease, and renal disease.[14]

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Multiple factors such as aging of PLHIV, smoking, dyslipidemic effects of HIV itself, and side effects of HAART such as dyslipidemia play major role in cardiovascular health.[15-18] Most of the ART classes cause some sort of dyslipidemia including protease inhibitors (Pis), non-nucleotide reverse transcriptase inhibitors (NNRTIs), and nucleotide reverse transcriptase inhibitors (NRTIs).[14] Evidence showed that the use of NNRTIs-based HAART was associated with a significant increase in total cholesterol (TC), low-density lipoprotein-C(LDL-C) as well as triglyceride (TG). Still, also there is 40% increase in high-density lipoprotein-C(HDL-C).[19] An Indian study showed significant changes in TC, HDL, and TG with the same regimen.[20] However, in MACS study (2001-2010) they showed the persistence of reduced HDL levels after the initiation of HAART.[21] Besides serum lipid profile, atherosclerosis and cardiovascular disease (CVD) risk are all known to vary by socio-demographic factors in the general population, and genetic polymorphisms involved in lipid metabolism may contribute to this variation.[21,22] Due to observed variation in ART-induced lipid abnormalities in different countries, it is essential to find out the nature of lipid abnormalities in these groups of patients under ART in Bangladesh to protect from the risk of CVD. No such report was available from any study in Bangladesh. Therefore, this study was designed to assess the impact of highly active antiretroviral therapy (HAAT) on fasting blood lipid profiles of HIV patients.

Methods

This prospective, observational study and was conducted in the ART Centre of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh, between January 2020 and June 2021. We included 49 HIV sero-positive patients (diagnosis based on rapid diagnostic test) attending ART Centre of Bangabandhu Sheikh Mujib Medical University (BSMMU) hospital.

The inclusion criteria were: i) patients aged ≥18 years old; ii) patients who are newly diagnosed with HIV before receiving any antiviral therapy; iii) patients who are able to attend at the selected health center (ART Center of BSMMU) on the day of data collection; iv) patients who were willing to participate in the study.

Our exclusion criteria were: i) pregnant women or breast-feeding mother, ii) patients with uncontrolled diabetes mellitus and BMI >25 kg/m2, iii) patients receiving any medication which may cause dyslipidaemia (e.g., ß-blockers, OCP, steroid, antidepressants, thiazide diuretics, retinoids etc.) iv) patients with any history acute illness (e.g., renal or pancreatic diseases, ischemic heart disease, chronic liver disease, etc.).

All data were recorded systematically in preformed data collection form. Quantitative data was expressed as mean and standard deviation, while qualitative data was expressed as frequency and percentage. Statistical analysis was performed by using Statistical Package for Social Sciences (SPSS) version 22.0 for windows. Paired sample t-test and McNemar's test were done for statistical analysis. Probability value <0.05 was considered as a level of significance.

The study was approved by Institutional Review Board of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh.

Results

Among 49 participants, almost half (49.0%) belonged to the ≤30 years age group. The mean age was 33.16±10.37 years with ranged from 19 to 60 years. More than two-thirds (69.4%) of the patients were

male and 14(28.6%) were female. 14(24.5%) had primary educational level, seconday 8(16.3%), higher secondary 9(18.4%) and 15 (30.6%) patients did graduate and above. In occupation, more than one-fourth 14(28.5%) of the patients were service holders, followed by 11(22.4%) housewives, 7(14.3%) students, 5(10.2%) unemployed, and 4(8.2%) business, while 8(16.3%) had other occupation. The majority (57.1%) of the patients were non-smokers, while 16.4% were ex-smokers, and 13(26.5%) were smokers (Table-I).

Table-I: Demographic profile of the participants (N=49)

Variables	Frequency	Percentage
Age group		
≤30 years	24	49.0
31-40 years	15	30.6
41-50 years	8	16.3
>50 years	2	4.1
Mean±SD	33.16±10.37 years	
Gender		
Male	34	69.4
Female	14	28.6
Transgender	1	2.0
Education		
Illiterate	3	6.1
Primary	14	28.6
Secondary	8	16.3
Higher secondary	9	18.4
Graduate and above	15	30.6
Occupation		
Student	7	14.3
Unemployed	5	10.2
Service	14	28.6
Business	4	8.2
Housewife	11	22.4
Others	8	16.3
Smoking status		
Smoker	13	26.5
Ex-smoker	8	16.3
Non-smoker	28	57.1

Regarding history of mode of transmission of HIV, nearly half (42.9%) of the patients had unprotected promiscuous sex, followed by commercial saloon users 19(38.8%), got from spouses 8(16.3%), blood transfusion 4(8.2%), sharing needles, syringes, or intravenous drug abuse 1(2%) and Unknown 7(14.3%) (Table-II). It was observed that nearly one-third (30.6%) of patients had WHO stage I followed by 14 (28.5%) stage II, 12 patients (24.5%) in stage III, and 8(16.3%) stage IV. Nearly half (46.9%) of patients had CDC Category A, followed by Category B 16(32.7%) and Category C 10(20.4%) (Table-III).

Table-II: Distribution of the study patients by transmission mode/risks (N=49)

Transmission mode /risks	Frequency	Percentage
Unprotected promiscuous sex	21	42.9
Commercial saloon user	19	38.8
Get from spouse	8	16.3
Blood transfusion	4	8.2
Sharing needles, syringes, or intravenous drug abuse	1	2.0
Vertical transmission	0	0.0
Unknown	7	14.3

Table-III: Distribution of the study patients by WHO stage and CDC category (N=49)

Variables	Frequency	Percentage
WHO stage		
Stage I	15	30.6
Stage II	14	28.6
Stage III	12	24.5
Stage IV	8	16.3
CDC category		
Category A	23	46.9
Category B	16	32.7
Category C	10	20.4

Regarding changes in lipid profile from baseline to follow-up (after 6 moths of HAART), it was observed that 3(9.7%) patients belonged to TC >200 mg/dl at baseline and after at least 6 months of HAART 6(19.4%) newly developed and 1(3.2%) was persistent. The majority (80.6%) of the patients belonged to HDL-C <40 mg/dl at baseline and after 6 months of HAART 3(9.7%) was newly developed and 16(51.6%) persisted to their baseline levels. 3(9.7%) patients belonged to LDL-C >130 mg/dl at baseline and after therapy, 2(6.5%) newly developed. More than one-third (35.5%) of patients belonged to TG >150 mg/dl at baseline and after therapy, 10(32.3%) newly developed and 9(29.0%) persisted to their baseline levels. The differences of HDL-C <40 and TG >150 were statistically significant (p<0.05) (Table-IV). Regarding dyslipidaemia, the majority (87.1%) of patients had dyslipidaemia before treatment and 26(83.9%) after HAART. The difference was statistically significant. It was observed that more than one-fourth (29.0%) of patients had dyslipidaemia stage II before treatment 10(32.3%) after HAART. The difference was statistically not significant (p>0.05) between the two groups. We found that the majority (80.6%) of patients had HDL-C (<40) before treatment 19(61.3%) after HAART. The difference was statistically significant (p<0.05) between the two groups (Table-V).

Table-IV: Comparing lipid profile at baseline and after 6 months of HAART (N=47)

Lipid Profile	At baseline Frequency (Percentage)	After at least 6 months of HAART			p-value
		Newly developed Frequency (Percentage)	Persistant Frequency (Percentage)	Total Frequency (Percentage)	
TC >200 (mg/dl)	3(9.7)	6(19.4)	1(3.2)	7(22.6)	0.214^{ns}
HDL <40 (mg/dl)	25(80.6)	3(9.7)	16(51.6)	19(61.3)	0.036^{s}
LDL >130 (mg/dl)	3(9.7)	2(6.5)	0(0.0)	2(6.5)	0.098^{ns}
TG >150 (mg/dl)	16(35.5)	10(32.3)	9(29.0)	19(61.3)	0.021^{s}

TC= Total cholesterol, HDL= High density lipoprotein, LDL= Low density lipoprotein, TG= Triglyceride; s= significant, ns= not significant.

Table-V: Lipid abnormalities and HDL-C of the study participants (N=31)

Variables	Before treatment		After HAART		p-value
	Frequency	Percentage	Frequency	Percentage	
Normal lipid profile	4	12.9	5	16.1	0.001^{s}
Dyslipidemia (Total)	27	87.1	26	83.9	
Stage I	9	29.0	8	25.8	0.982^{ns}
Stage II	9	29.0	10	32.3	
Stage III	7	22.6	6	19.4	
Stage IV	2	6.4	2	6.4	
HDL-C (<40)	25	80.6	19	61.3	0.031^{s}
HDL-C (>40)	6	19.4	12	38.7	

HDL-C- High density lipoprotein- cholesterol; s= significant, ns=not significant.

Discussion

In this study, most patients (49.0%) were ≤30 years old and the mean age was 33.16±10.37 years varying from 19 to 60 years. Similar findings were reported by Tilahun *et al*., as the mean age of study participants was 35.31±7.20 years.[22] In another study, the mean age was 36 years, which supports the present study.[20] In our study, we observed that 69.4% of patients were male, 28.6% female, and 2.0% transgender. Similar finding was reported by an Indian study, as 79% were male and the rest 21% were female,[20] while others found female predominance in their respective studies, where they found 54.82% and 75.6% were female respectively.[22,23]

In this current study, it was observed that 24.5% of patients had educational level primary, and more than one-fourth of patients did higher studies (30.6%), SSC (16.3%), and HSC (18.4%). Tilahun *et al.* reported that the majority of patients can read and write.[22] In this present study it was observed that 57.1% patients were non smoker, 16.4% ex- smoker and 26.5% smoker. Tilahun *et al*. reported that 12.8% of the patients had positive history of smoking, which is much lower than our finding.[22]

Regarding the transmission mode/risks in this current study, it was observed that 42.9% of patients had unprotected promiscuous sex, followed by 38.8% use of commercial saloon for shaving/haircut, getting from spouse (16.3%), blood transfusion (8.2%), sharing needles, syringes and intravenous drug abuse 2%, 0% by vertical transmission and unknown (14.3%). Pujari *et al.* mentioned ithat 94.0% had acquired HIV infection by heterosexual transmission in India.[24]

In this present study, we observed that at the time of diagnosis majority of patients (30.6%) had WHO stage I followed by 28.5% in stage II, 24.5% in stage III, and 16.3% in stage IV. Pujari *et al*. observed that at the initiation of therapy 38.6% and 36.6% of patients had WHO stage IV disease respectively.[24]

In this current study, it was observed that 9.7% had TC ≥200. The majority 80.6% patients belonged to HDL-C <40 (mg/dl) and the mean was 31.4±9.26 (gm/dl). Regarding LDL-C, 9.7% of patients had LDL-C (>130 mg/dl), and the mean was 85.34±28.79 (gm/dl). About half 51.6% of patients belonged to TG >150 (mg/dl) and the mean was 154.46±59.62 (gm/dl). Tilahun *et al.* observed that 39.9% of the study participants had total cholesterol of ≥200mg/dl, 39.9% had diminished levels of HDL-C (<40 mg/dl), 38.1% had elevated levels of LDL-C (>130 mg/dl) and 49.1% had elevated level of TG (>150 mg/dl).[22] At baseline, Padmapriyadarsini *et al.* found that TG level was >150 mg/dL in 31% of patients, TC level was 200 mg/dL in 2% patients, HDL-C was 40 mg/dL in 91% of patients, LDL-C was 130 mg/dL in 3% patients. They observed that, at baseline, total cholesterol, LDL-C, and HDL-C levels were low, however, triglyceride (TG) levels were in the regular range, which is comparable with the current study.[20]

Sarkar & Brown observed in their study that HIV infection is associated with dyslipidemia.[25] In the absence of antiretroviral therapy (ART), HIV infection results in lower total cholesterol (TC), high-density lipoprotein (HDL-C), and low-density lipoprotein (LDL-C) levels.[26] Grunfeld *et al.* found that participants with AIDS had the highest levels of TG compared to participants with HIV and controls. TC and HDL-C were lower in individuals with AIDS and in those with HIV compared to controls, and LDL-C was significantly lower in individuals with AIDS compared to control participants.[27] The high levels of TG in participants with AIDS are secondary to increased

hepatic output of VLDL and slower TG clearance.[27] In this current study, it was observed that the mean TG & HDL were significantly ($p<0.05$) increased at follow up but TC and LDL were almost alike between baseline and follow-up. Padmapriyadarsini *et al.* observed after 6 months of ART, significant increases were in TC level (125 vs 174mg/dL; $p<0.001$), LDL-c level (83 vs 112 mg/dL; $p<0.001$), and HDL-c level (26 vs 44 mg/dL; $p<0.001$), which differ with the present study.[20]

In this present study, it was observed that 87.1% of patients developed dyslipidemia before treatment and 83.9% after HAART. Development of dyslipidemia was significantly ($p<0.05$) revealed after HAART. Tilahun *et al.* showed the prevalence of dyslipidemia in their study was 63.60%, of them 73.68% were on HAART and 53.51% were HAART naïve HIV positive clients.[22] Melzi *et al.* mentioned that HIV-1 infection causes a specific pattern of dyslipidemia, resulting from a combination of increased production and decreased clearance of lipoproteins.[28]

In this present study, it was observed that 80.6% of patients had HDL-C (<40) before treatment and 61.3% in after HAART. HDL-C (<40) level significantly declined after HAART. Mulu et al. (2021) study showed that the prevalence of HDL-C <40mg/dl among HIV clients on HAART was 43.8%.[22] Similar prevalence was also reported by Tadewos *et al*., which was 43.4%.[29] The HDL-C level was unaffected by HAART status as reported by Nsagha *et al.*[23] However, the prevalence of HDL-c <40mg/dl among HIV clients on HAART is higher in our study. However, Pujari *et al.* found that an 18-month treatment with line ART regimens was associated with a significant increase in HDL-c level, which supports the present study.[24]

Our study was a single-center study. We took a small sample size due to our short study period. The study population was selected from one selected ART center in Dhaka city, so the results of the study may not reflect the exact picture of the country. We did not follow-up with our patients for more than 6 months and do not know other possible interference that may happen in the long term with them.

Conclusion

We found that unprotected promiscuous sex was the most common mode of transmission of HIV. Triglyceride levels were significantly elevated after HAART, but TC, HDL-C, and LDL-C levels almost remained the same between baseline and after HAART. Development of dyslipidaemia was found more than two-thirds (83.9%) after HAART. HIV-infected patients receiving WHO-recommended1 first-line HAART have a high prevalence of lipid profile derangement. Therefore, further studies with a prospective and longitudinal study design including larger sample size need to be done to evaluate the long-term effects of HAART treatment on lipid profiles of HIV patients.

References

1. *UNAIDS. Global HIV & AIDS statistics – Fact sheet [Internet]. UNAIDS. Available from: https://www.unaids.org/en/resources/fact-sheet (Accessed November 24, 2021).*
2. *HIV/AIDS Data Hub for the Asia Pacific.. Bangladesh. Available from: https://www.aidsdatahub.org/country-profiles/bangladesh (Accessed November 25, 2021).*
3. *Lohse N, Hansen A, Pedersen G, Kronbora G, Gerstoft J, Sorensen H, et al. Survival of Persons with and without HIV Infection in Denmark, 1995–2005. Ann Int Med. 1995;146(2):87-95.*

4. *Grinsztejn B, Luz PM, Pacheco AG, Santos DVG, Velasque L, Moreira RI, et al. Changing mortality profile among HIV-infected patients in Rio de Janeiro, Brazil: shifting from AIDS to non-AIDS related conditions in the HAART era. PLoS One. 2013;8(4):e59768.*
5. *Palella FJ, Delaney KM, Moorman AC et al. Declining morbidity and mortality among patients with advanced human immunodeficiency virus infection. HIV Outpatient Study Investigators. N Engl J Med .1998;338:853-60.*
6. *Mocroft A, Vella S, Benfield TL, Chiesi A, Miller V, Gargalianos P, et al. Changing patterns of mortality across Europe in patients infected with HIV-1. EuroSida Study Group. Lancet 1998;352(9142):1725-30.*
7. *Carr A, Cooper DA. Adverse effects of antiretroviral therapy. Lancet 2000;356:1423-30.*
8. *Hicks C, Currier J, Sax P, Sherer R, Wanke C. Current management challenges in HIV: tolerability of antiretrovirals and metabolic complications. AIDS Patient Care STDs 2003;17:221-33.*
9. *Currier JS. Cardiovascular risk associated with HIV therapy. J Acquir Immune Defic Syndr 2002;31:S16-32.*
10. *Rhew DC, Bernal M, Aguilar D, Iloeje U, Goetz MB. Association between protease inhibitor use and increased cardiovascular risk in patients infected with human immunodeficiency virus: a systematic review. Clin Infect Dis 2003;37:959-72.*
11. *Carr A, Carr A, Samaras K, Burton S, Law M, Freund J, et al. A syndrome of peripheral lipodystrophy, hyperlipidaemia and insulin resistance in patients receiving HIV protease inhibitors. AIDS. 1998;12(7):F51-8.*
12. *Tsiodras S, Mantzoros C, Hammer S, Samore M. Effects of protease inhibitors on hyperglycemia, hyperlipidemia, and lipodystrophy. A 5-year cohort study. Arch Intern Med. 2000;160:2050-6.*
13. *Palacios R, Santos J, Ruiz J, Gonzalez M, Marquez M. Factors associated with the development of diabetes in HIV-infected patients on antiretroviral therapy: a case-control study. AIDS. 2003;17:933-35.*
14. *Feeney ER, Mallon PW. HIV and HAART-associated dyslipidemia. Open Cardiovasc Med J. 2011;5:49.*
15. *Mack KA, Ory MG. AIDS and older Americans at the end of the twentieth century. J Acquir Immune Defic Syndr. 2003;33:S68-75.*
16. *Mamary EM, Bahrs D, Martinez S. Cigarette smoking and the desire to quit among individuals living with HIV. AIDS Patient Care STDs. 2002;16(1):39-42.*
17. *Grunfeld C, Kotler DP, Hamadeh R, Tierney A, Wang J, Pierson Jr RN. Hypertriglyceridemia in the acquired immunodeficiency syndrome. Am J Med. 1989;86(1):27-31.*
18. *Grover SA, Coupal L, Gilmore N, Mukherjee J. Impact of dyslipidemia associated with Highly Active Antiretroviral Therapy (HAART) on cardiovascular risk and life expectancy. Am J Cardiol. 2005;95(5):586-91.*
19. *Haubrich RH, Riddler SA, DiRienzo AG, Komarow L, Powderly WG, Klingman K, et al. Metabolic outcomes in a randomized trial of nucleoside, nonnucleoside and protease inhibitor-sparing regimens for initial HIV treatment. AIDS. 2009;23(9):1109-18.*
20. *Padmapriyadarsini C, Ramesh Kumar S, Terrin N, Narendran G, Menon PA, Ramachandran G, et al. Dyslipidemia among HIV-infected Patients with tuberculosis taking once-daily nonnucleoside reverse-transcriptase inhibitor-based antiretroviral therapy in India. Clin Infect Dis. 2011;52(4):540-6.*
21. *Nicholaou MJ, Martinson JJ, Abraham AG, Brown TT, Hussain SK, Wolinsky SM, et al. HAART-associated dyslipidemia varies by biogeographical ancestry in the multicenter AIDS cohort study. AIDS Res Hum Retroviruses. 2013;29(6):871-9.*
22. *Tilahun A, Chekol E, Teklemaryam AB, Agidew MM, Tilahun Z, Admassu FT, et al. Prevalence and predictors of dyslipidemia among HAART treated and HAART naive HIV positive clients attending Debre Tabor Hospital, Debre Tabor, Ethiopia. Heliyon. 2022;8(11):e11342.*

23. *Nsagha DS, Weledji EP, Assob NJ, Njunda LA, Tanue EA, Kibu OD, et al. Highly active antiretroviral therapy and dyslipidemia in people living with HIV/AIDS in Fako Division, South West Region of Cameroon. BMC Cardiovasc Disord. 2015;15:95.*

24. *Pujari SN, Dravid A, Naik E, Bhagat S, Tash K, Nadler JP, Sinnott JT. Lipodystrophy and dyslipidemia among patients taking first-line, World Health Organization-recommended highly active antiretroviral therapy regimens in Western India. J Acquir Immune Defic Syndr. 2005;39(2):199-202.*

25. *Sarkar S, Brown TT. Lipid disorders in people with HIV. In: Feingold KR, Ahmed SF, Anawalt B, et al. eds. Endotext [Internet]. South Dartmouth, MA: MDText.com, Inc.; 2000.*

26. *Riddler SA, Smit E, Cole SR, Li R, Chmiel JS, Dobs A, et al. Impact of HIV infection and HAART on serum lipids in men. JAMA. 2003;289(22):2978-82.*

27. *Grunfeld C, Pang MI, Doerrler W, Shigenaga JK, Jensen P, Feingold KR. Lipids, lipoproteins, triglyceride clearance, and cytokines in human immunodeficiency virus infection and the acquired immunodeficiency syndrome. J Clin Endocrinol Metab. 1992;74(5):1045-52.*

28. *Melzi S, Carenzi L, Cossu MV, Passerini S, Capetti A, Rizzardini G. Lipid metabolism and cardiovascular risk in HIV-1 infection and HAART: present and future problems. Cholesterol. 2010;2010:271504.*

29. *Tadewos A, Addis Z, Ambachew H, Banerjee S. Prevalence of dyslipidemia among HIV-infected patients using first-line highly active antiretroviral therapy in Southern Ethiopia: a cross-sectional comparative group study. AIDS Res Ther. 2012;9(1):31.*