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Changes in the lipid profile in people with HIV after one year of antiretroviral therapy – the significance of immune parameters

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ABSTRACT

Objectives: This study aimed to analyze lipid profiles among people with HIV and observe changes in lipid parameters during 1 year of antiretroviral therapy (ART), with particular reference to immune parameters.
Methods: We analyzed adult newly diagnosed people with HIV (PWH) who started ART, continued uninterruptedly for 1 year and achieved complete viral suppression. Patients were not receiving lipid-lowering therapy. The cluster of differentiation (CD4) count, CD4:CD8 ratio, HIV type 1 viral load, and lipid profile were examined at HIV diagnosis and after 12 months of ART.
Results: The study included 70 patients. Significant increases in total, high-density lipoprotein (HDL), and low-density lipoprotein (LDL) cholesterol and decreases in triglyceride concentrations after 1 year of ART were observed. A baseline CD4 count <200/μl was associated with higher baseline LDL cholesterol ($P = 0.036$), and female sex with elevated total, LDL, and non-HDL cholesterol ($P = 0.005$; $P = 0.011$; $P = 0.008$). Patients with baseline CD4 counts <200/μl had significantly higher total, LDL, and non-HDL cholesterol ($P = 0.033$; $P = 0.009$; $P = 0.009$) and triglyceride ($P = 0.003$) levels after 1 year of ART than patients with CD4 levels ≥200/μl.
Conclusions: Lipid parameters should be regularly assessed in all PWH receiving ART, especially in patients with baseline CD4 counts <200/μl.

Introduction

HIV, regardless of the stage of infection, is a known risk factor for altered lipid profiles. People with HIV (PWH) often experience low concentrations of high-density lipoprotein (HDL) cholesterol and hypertriglyceridemia, which are both considered high-risk lipid profiles for atherosclerosis and cardiovascular disease (CVD). The reasons for this phenomenon are multivariate, and it is suggested that HIV may be associated with endothelial dysfunction [1]. Moreover, circulating HIV has a proinflammatory effect by promoting the expression of adhesive proteins and cytokines [2]. Another possible but not well-examined cause of impaired lipid profiles may be the impact of altered immune parameters in PWH.

Untreated HIV infection gradually leads to depletion of the cluster of differentiation (CD4) lymphocyte T-cell count and a decrease in the CD4:CD8 ratio. While the relationship of the decline in immune parameters with the development of opportunistic infections and AIDS is well known, its impact on the lipid profile remains unclear. Studies suggest that a low CD4 lymphocyte count should be considered unfavorable in terms of the lipid profile. Compared with PWH with higher CD4 cell counts, those with lower lymphocyte counts are more likely to have low HDL cholesterol concentrations and higher triglyceride levels [2]. Furthermore, a history of AIDS-related events, which depend on a decrease in the CD4 lymphocyte count, was also reported to be associated with increased total cholesterol and triglyceride concentrations [3]. The crucial factor impacting both immune parameters and lipid profiles in PWH is antiretroviral therapy (ART), whose role in changing the natural course of HIV infection is tremendous, but its cardiovascular effects are ambiguous.

Novel antiretroviral agents are known to have high effectiveness and low toxicity [4]. With increasing life expectancy of PWH, an increase in cardiovascular effects has been reported, including the worsening of lipid parameters in comparison with those in the general population. Compared with ART-naïve PWH, PWH receiving ART have higher concentrations of total cholesterol, low-density lipoprotein (LDL) cholesterol and triglycerides [5]. The application scheme of ART is also highly important since not all antiretroviral agents have the same potential to cause dyslipidemia. Protease inhibitors (PIs) are a class of ARTs that are considered unfavorable in terms of their lipid profile [6]. Among other agents, tenofovir alafenamide (TAF) may also be associated with in-

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Table 1
Study population characteristics.

	Baseline	After 1 year of antiretroviral therapy	*P*
Total cholesterol (mmol/l) mean (SD)	4.15 (1.16)	4.81 (1.08)	**<0.001**
High-density lipoprotein cholesterol (mmol/l) mean (SD)	0.84 (0.36)	1.22 (0.43)	**<0.001**
Low-density lipoprotein cholesterol (mmol/l) mean (SD)	2.46 (0.43)	2.79 (0.86)	**0.007**
Non-high-density lipoprotein cholesterol (mmol/l) mean (SD)	3.31 (1.02)	3.60 (1.05)	**0.028**
Triglyceride (mmol/l) mean (SD)	1.98 (0.94)	1.82 (1.13)	0.285
Atherogenic index of plasma mean (SD)	0.37 (0.33)	0.13 (0.33)	**<0.001**
CD4 lymphocyte count (cells/μl) mean (SD)	201.74 (273.39)	407.29 (290.25)	**<0.001**
CD4:CD8 ratio mean (SD)	0.26 (0.30)	0.56 (0.44)	**<0.001**

CD, clusters of differentiation.

creases in total and LDL cholesterol [7]. In contrast, some regimens may be beneficial in terms of lipid profile: after switching to doravirine from different regimens, decreases in total and LDL cholesterol and triglycerides have been observed [8].

Various other factors may impact the lipid profile of PWH. Among PWH, female sex is associated with higher total, LDL and non-HDL cholesterol levels and higher triglyceride concentrations [9]. Moreover, chronic hepatitis C virus (HCV) and hepatitis B virus (HBV) infections can influence the lipid profile in PWH. Studies have shown that PWH coinfected with HCV have lower total cholesterol, LDL cholesterol, and HDL cholesterol levels than PWH infection alone [10]. Patients with HIV/HBV coinfection also seem to have a lower risk of dyslipidemia than PWH do [11].

Our study aimed to evaluate the lipid profile among people with newly diagnosed HIV infection who were not previously exposed to ART and to observe the change in the lipid profile after 1 year of effective ART. This study focused mostly on the impact of the application scheme of ART and the effects on the CD4 lymphocyte count and CD4:CD8 ratio in the context of lipid parameters.

Materials and methods

Study design

We conducted an observational cohort study evaluating changes in the lipid profile of PWH before the implementation of antiretroviral treatment and after 1 year of persistent ART. We included adult (age 18 or older) patients with newly diagnosed HIV infection with no previous history of ART who started ART, continued uninterruptedly for a minimum of 1 year, and reached complete viral suppression at a follow-up visit after 1 year since the initiation of ART. All analyzed patients were hospitalized in our department between 2016 and 2023. The exclusion criteria were a history of dyslipidemia, receiving lipid-lowering therapy, or a change in the ART scheme during the analyzed period.

Assessments

All patients were analyzed twice—before the implementation of ART and after 1 year of persistent treatment. At the starting and ending point of the study, all patients were subjected to physical examination and laboratory testing. The scheme of applied ART during the observation period was analyzed. The modification of any medication or the form of medication was considered a change in the ART scheme. Blood samples were collected twice—before the initiation of ART and after 1 year of persistent treatment. The CD4 count, CD4:CD8 ratio, HIV viral load, total cholesterol, LDL cholesterol, HDL cholesterol and triglyceride levels were repeatedly analyzed. The non-HDL cholesterol concentration and the atherogenic index of plasma (AIP) were measured. The immunophenotyping analyses, including measurements of the absolute counts of the T lymphocyte (CD3+) subsets, namely, the CD4+ (helper/inducer), CD8+ (suppressor/cytotoxic), and CD4:CD8 ratios, were performed via flow cytometry with three-color direct immunofluorescence reagents—TriTEST^{TM} (BD Biosciences, Australia). The HIV viral load was assessed via the Abbott real-time HIV type 1 assay via an *in vitro* reverse transcription-polymerase chain reaction assay with homogenous real-time fluorescent detection for the quantification of HIV type 1 in the automated m2000 system in human plasma. An HIV viral load <50 copies/mL was considered undetectable. All patients were assessed in terms of HCV and HBV coinfections. HCV coinfection was confirmed by polymerase chain reaction of HCV RNA, and HBV infection was confirmed by HBs antigen. The data contained in the boxplots are reported as the mean value and standard deviation.

Statistical analysis

The Shapiro–Wilk test was performed to verify the normality of the distributions of the analyzed variables. Student's *t*-test or the Mann-Whitney U test was used to evaluate the difference in the mean values of the analyzed variables. Paired *t*-test or Wilcoxon signed-rank test was used for the analysis of the differences in mean values of analyzed parameters before ART implementation and after 1 year of ART. ANOVA was used to assess the difference in the mean value among more than two quantitative variables. The Tukey Honestly Significant Difference (HSD) test was used as a *post hoc* test. Pearson correlation was performed to measure the correlation between two sets of data. Multiple linear regression was performed with adjustments for age, sex, HCV, and HBV coinfections, and the application scheme of ART. The *P*-value was set at 0.05. All the statistical analyses were performed via Python 3.7 software and the Statistica 13.1 program (StatSoft Poland, Kraków, Poland).

Results

Study population

We analyzed a population of 70 patients (58 men and 12 women) who were diagnosed with HIV infection and had no previous history of ART. The mean age of the analyzed patients was 38.1 years (standard deviation 10.81). Among our patients, seven were diagnosed with chronic HCV infection (five men, two women), and four were diagnosed with chronic HBV infection (four men, zero women). There were no patients with HIV/HCV/HBV coinfection. The basic characteristics of the study group are presented in Table 1.

CD4 and lipid profiles

We analyzed whether a lower CD4 count before ART implementation was associated with a worse baseline lipid profile. The results are presented in Table 2 and Figure 1.

We did not observe statistically significant differences in total cholesterol, HDL cholesterol, LDL cholesterol, non-HDL cholesterol, triglyceride concentration, or AIP between patients with baseline CD4 counts <200/μl and those with baseline CD4 counts ≥500/μl ($P = 0.443$, $P = 0.414$, $P = 0.605$, $P = 0.365$, $P = 0.486$, $P = 0.783$, respectively). The results are shown in Figure 1.

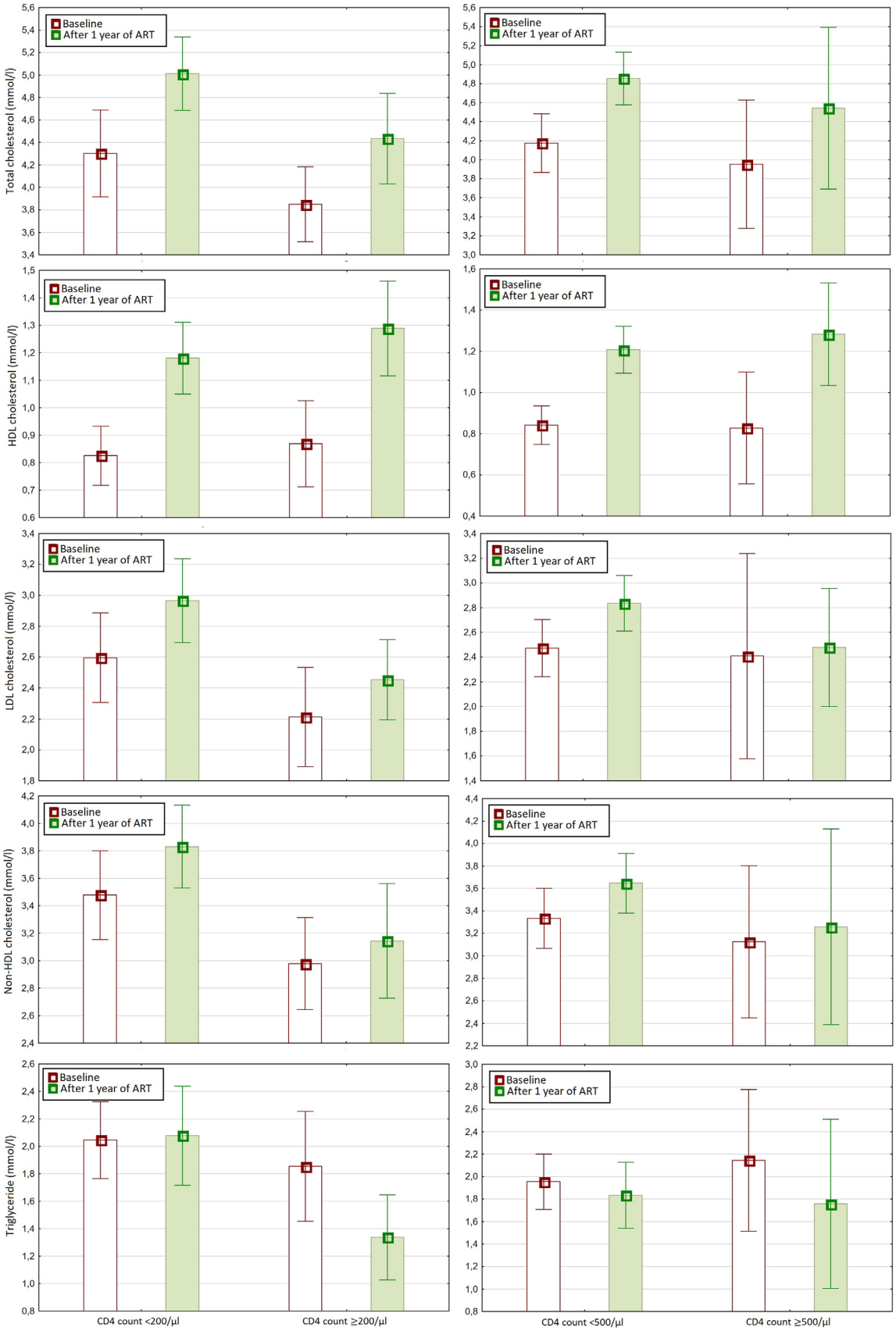

Figure 1. The comparison of baseline and 1-year post-treatment implementation lipid profile among people with HIV with different baseline CD4 cell counts. ART, antiretroviral therapy; CD, cluster of differentiation; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Table 2
Comparison of the baseline lipid profiles of people with HIV with different CD4 counts.

	Baseline CD4 <200 (n = 46)	Baseline CD4 ≥200 (n = 24)	*P*	Baseline CD4 <500 (n = 61)	Baseline CD4 ≥500 (n = 9)	*P*
Total cholesterol (mmol/l) mean (SD)	4.30 (1.30)	3.85 (0.79)	0.122	4.18 (1.20)	3.95 (0.88)	0.595
High-density lipoprotein cholesterol (mmol/l) mean (SD)	0.83 (0.36)	0.87 (0.37)	0.252	0.84 (0.37)	0.83 (0.35)	0.493
Low-density lipoprotein cholesterol (mmol/l) mean (SD)	2.60 (0.97)	2.21 (0.76)	**0.036**	2.47 (0.90)	2.41 (1.08)	0.846
Non-high-density lipoprotein cholesterol (mmol/l) mean (SD)	3.48 (1.09)	2.98 (0.79)	0.052	3.33 (1.04)	3.12 (0.88)	0.281
Triglyceride (mmol/l) mean (SD)	2.05 (0.94)	1.85 (0.95)	0.087	1.96 (0.96)	2.14 (0.82)	0.299
Atherogenic index of plasma mean (SD)	0.39 (0.32)	0.32 (0.34)	0.402	0.36 (0.34)	0.42 (0.27)	0.591

CD, clusters of differentiation.

Table 3
Comparison of 1-year post-antiretroviral therapy implementation lipid profiles in people with HIV with different baseline CD4 counts.

	Baseline CD4 lymphocyte count <200 cells/μl (n = 46)	Baseline CD4 lymphocyte count ≥200 cells/μl (n = 24)	*P*
Total cholesterol (mmol/l) mean (SD)	5.01 (1.10)	4.43 (0.95)	**0.033**
High-density lipoprotein cholesterol (mmol/l) mean (SD)	1.18 (0.44)	1.29 (0.41)	0.138
Low-density lipoprotein cholesterol (mmol/l) mean (SD)	2.97 (0.92)	2.45 (0.62)	**0.009**
Non-high-density lipoprotein cholesterol (mmol/l) mean (SD)	3.83 (1.02)	3.14 (0.99)	**0.009**
Triglyceride (mmol/l) mean (SD)	2.08 (1.21)	1.34 (0.73)	**0.003**
Atherogenic index of plasma mean (SD)	0.21 (0.34)	−0.01 (0.27)	**0.008**

CD, clusters of differentiation.

Table 4
The correlation between CD4 count and CD4:CD8 ratio growth during 1 year of antiretroviral therapy and the change in lipid profile concentrations.

	CD4 univariate correlation coefficient	Univariate *P*	Multiple linear regression *P*	CD4:CD8 univariate correlation coefficient	Univariate *P*	Multiple linear regression *P*
Total cholesterol (mmol/l) mean (SD)	0.056	0.647	0.970	−0.150	0.216	0.282
High-density lipoprotein cholesterol (mmol/l) mean (SD)	0.018	0.880	0.975	0.069	0.571	0.317
Low-density lipoprotein cholesterol (mmol/l) mean (SD)	0.043	0.724	0,980	−0.074	0.542	0.263
Non-high-density lipoprotein cholesterol (mmol/l) mean (SD)	0.057	0.641	0.979	−0.197	0.103	0.317
Triglyceride (mmol/l) mean (SD)	−0.075	0.536	0.978	−0.268	**0.025**	0.227
Atherogenic index of plasma mean (SD)	−0.065	0.594	0.979	−0.194	0.108	0.328

CD, clusters of differentiation.

Table 5
Lipid profile differences among men and women before the initiation of antiretroviral therapy.

	Women (n = 12)	Men (n = 58)	*P*
Total cholesterol (mmol/l) mean (SD)	5.00 (1.22)	3.97 (1.08)	**0.005**
High-density lipoprotein cholesterol (mmol/l) mean (SD)	1.00 (0.38)	0.81 (0.35)	0.061
Low-density lipoprotein cholesterol (mmol/l) mean (SD)	3.07 (1.05)	2.34 (0.85)	**0.011**
Non-high-density lipoprotein cholesterol (mmol/l) mean (SD)	4.00 (1.08)	3.16 (0.95)	**0.008**
Triglyceride (mmol/l) mean (SD)	2.36 (1.12)	1.90 (0.89)	0.086
Atherogenic index of plasma mean (SD)	0.36 (0.30)	0.37 (0.33)	0.899

CD, clusters of differentiation.

Table 3 presents the lipid profile after 1 year of ART in patients with baseline CD4 lymphocyte counts <200 cells/μl and ≥200 cells/μl.

We analyzed the correlation of the CD4 count and CD4:CD8 ratio growth with the total cholesterol, HDL cholesterol, LDL cholesterol, non-HDL cholesterol, triglyceride, and AIP changes after 1 year of ART. The results are presented in Table 4.

ART and lipid profile

All of the analyzed patients received ART consisting of one or two nucleoside reverse transcriptase inhibitors (NRTI) plus one integrase strand transfer inhibitor (InSTI) (56 patients), one PI (seven patients), or one non-nucleoside reverse transcriptase inhibitor (NNRTI) (seven patients). We analyzed whether receiving InSTI/PI/NNRTI for 1 year was associated with a worsening of the lipid profile, and we did not observe statistically significant differences in the changes in total cholesterol, HDL cholesterol, LDL cholesterol, non-HDL cholesterol, triglyceride concentration, or AIP ($P = 0.711$, $P = 0.780$, $P = 0.803$, $P = 0.559$, $P = 0.198$, $P = 0.298$, respectively).

TAF/ tenofovir disoproxil (TDF)

In the analyzed group of patients, 18 individuals received TDF, and 49 people received TAF. We did not observe statistically significant differences in total cholesterol, HDL cholesterol, LDL cholesterol, non-HDL cholesterol, triglyceride concentration, or AIP among people treated with TDF or TAF after 1 year of antiretroviral treatment ($P = 0.396$, $P = 0.885$, $P = 0.291$, $P = 0.298$, $P = 0.381$, $P = 0.911$, respectively).

Bictegravir (BIC)/ dolutegravir (DTG)/ elvitegravir (EVG)

Among patients receiving InSTI-based ART, 22 individuals received BIC, 14 received DTG, and 18 received elvitegravir (boosted with co-

bicistat). We aimed to investigate whether treatment with individual medications was associated with significantly different lipid profiles after 1 year of therapy. The results are presented in Figure 2.

We observed statistically significant differences in LDL cholesterol concentration among the three analyzed groups. Therefore, we performed a *post hoc* Tukey HSD test, which revealed that LDL cholesterol concentration growth was significantly greater among people receiving elvitegravir boosted with cobicistat (EVG/c) than among people receiving BIC (P = 0.004). The test revealed no statistically significant difference in LDL growth among people who received a BIC vs DTG (P = 0.385) or DTG vs EVG/c (P = 0.062). However, individuals receiving EVG/c-based therapy had significantly lower baseline LDL cholesterol concentrations (2.07 mmol/l) than patients receiving BIC-based therapy did (2.89 mmol/l) (P = 0.001), and the mean LDL cholesterol concentration after 1 year of receiving EVG/c-based therapy and BIC-based therapy was insignificant (2.87 for EGV/c-based therapy and 2.83 mmol/l for BIC-based therapy, P = 0.478).

Sex

We analyzed whether lipid profiles differed among men and women before ART implementation. The results are presented in Table 5.

We did not observe statistically significant differences in the changes in total cholesterol, HDL cholesterol, LDL cholesterol, non-HDL cholesterol, triglyceride concentration, or AIP among men and women after 1 year of antiretroviral treatment (P = 0.164, P = 0.429, P = 0.079, P = 0.108, P = 0.715, P = 0.956, respectively).

HCV/HBV coinfection

Owing to the small number of patients with HCV infection and HBV infection, we analyzed patients with HCV or HBV coinfection together and compared the results to those in a population of individuals with HIV mono-infection. We did not observe statistically significant differences in total cholesterol, HDL cholesterol, LDL cholesterol, non-HDL cholesterol, triglyceride concentration or the AIP before the introduction of ART among individuals with HCV or HBV chronic coinfection and those with HIV mono-infection (P = 0.939, P = 0.433, P = 0.118, P = 0.974, P = 0.484, P = 0.854, respectively) or after 1 year of treatment (P = 0.355, P = 0.610, P = 0.210, P = 0.430, P = 0.223, P = 0.362, respectively).

Discussion

Our study aimed to evaluate the importance of immune parameters in PWH in relation to the concentration of lipid parameters. We chose a group of newly diagnosed antiretroviral-naïve patients not receiving lipid-lowering therapy and observed alterations in the CD4 cell count, CD4:CD8 ratio, and lipid profile during 1 year of uninterrupted ART. At the time of HIV diagnosis, the study population's mean LDL cholesterol, non-HDL cholesterol and triglyceride concentrations were elevated; however, the patients did not have CVD or a 10-year CVD risk ≥10%; therefore, lipid-lowering therapy was not introduced, and all patients were advised to have lifestyle modifications, according to the European AIDS Society guidelines 2022 [12].

After 1 year of follow-up, we observed statistically significant increases in total cholesterol, LDL cholesterol, HDL cholesterol, and non-HDL cholesterol in comparison with the baseline lipid profile. Interestingly, we observed a decrease in the triglyceride concentration, however, that difference was not statistically significant. The obtained data regarding the growth in total and LDL cholesterol during ART are generally consistent with current research [13]. However, the change in triglyceride concentration in ART-treated patients is ambiguous. Some studies suggest an increase in the triglyceride concentration after the implementation of ART, which is different than that reported in our

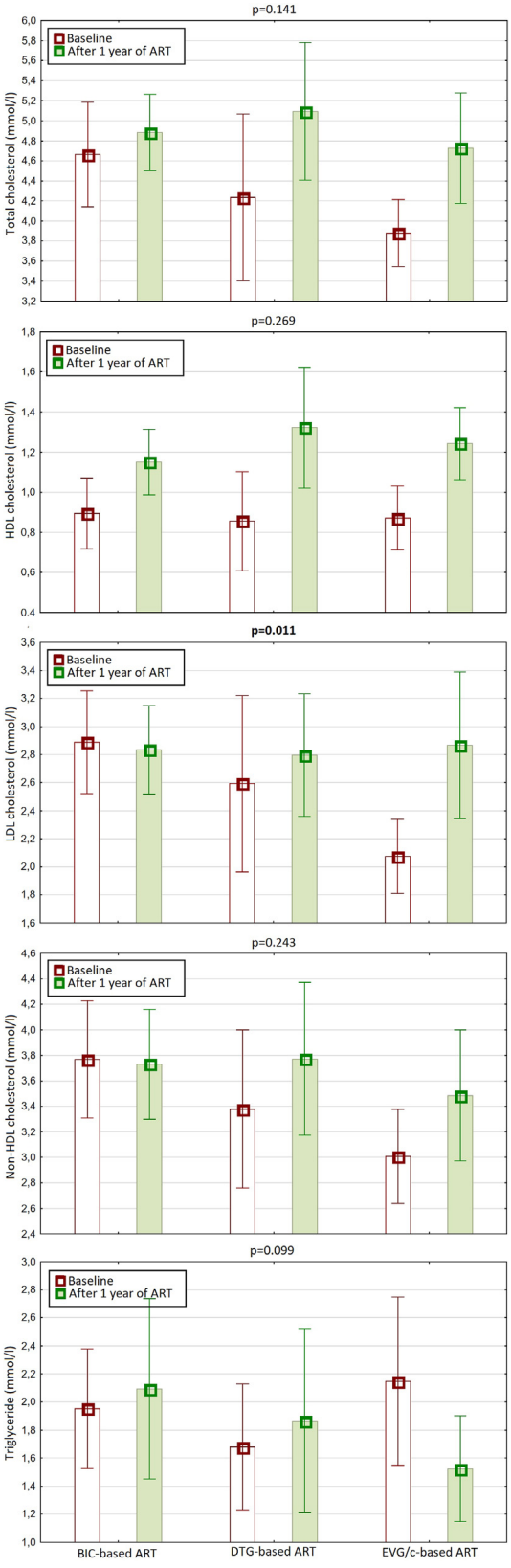

Figure 2. The comparison of baseline and 1-year post-treatment implementation lipid profile among people with HIV receiving integrase strand transfer inhibitor-based therapy.
BIC, bictegravir; DTG, dolutegravir; EVG/c, elvitegravir boosted with cobicistat; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

population [5]. On the other hand, the triglyceride concentration may also decrease depending on the ART regimen [14].

The AIP correlates with the development of cardiovascular events and HIV is known to increase it [15]. However, there are limited data on the impact of ART on AIP. In our study, despite the general worsening of the lipid profile after the initiation of ART, we observed a significant decrease in the AIP score after 1 year of treatment. Since the AIP may be an independent factor impacting the risk of CVD in PWH, it may be possible that with the use of ART, cardiovascular risk may be reduced, regardless of the lipid profile [16].

ART is currently characterized by high safety, high effectiveness, and low toxicity; however, people receiving ART may be at increased risk of dyslipidemia [5]. In our study, we did not observe statistically significant differences in lipid concentrations among people receiving InSTI/PI/NNRTI-based regimens; however, 80% of the individuals included in our study were receiving InSTI. Studies have shown that InSTIs are associated with a lower risk of dyslipidemia than PIs and NNRTIs [17]. Our patients were receiving BIC, DTG, and EVG/c, and we observed greater LDL cholesterol growth among people receiving EVG/c than among people receiving BIC. The results may be biased because individuals receiving EVG/c-based therapy had significantly lower baseline LDL cholesterol. However, the results are consistent with those of randomized controlled trials, which also suggest that treatment with EVG/c is associated with worse lipid profiles than BIC- or DTG-based therapy [18]. Another drug associated with an unfavorable lipid profile is TAF, which is especially associated with increases in LDL cholesterol and triglyceride levels after switching from regimens containing TDF [7]. In our study, we did not observe significant differences in lipid concentrations among people receiving TAF and those receiving TDF. However, this may be due to the small population of individuals receiving TDF (18 patients) in comparison to those receiving TAF (49 patients).

Our study aimed to evaluate the impact of immune parameters on the lipid profile. First, we analyzed whether individuals with lower baseline CD4 counts and CD4:CD8 ratios had more unfavorable baseline lipid profiles. We observed that a lower CD4 count and CD4:CD8 ratio were associated with higher concentrations of total cholesterol, LDL cholesterol and non-HDL cholesterol. However, only LDL cholesterol was significantly higher among people with a baseline CD4 count <200/μl than among PWH with a CD4 count ≥200/μl. There is not much current research evaluating the impact of the CD4 count on the lipid profile; however, our findings seem to be consistent with the established statement that a lower CD4 count is generally considered a predictor of dyslipidemia [19]. On the other hand, research has also suggested that dyslipidemia in PWH with low CD4 counts is associated mostly with increases in very low-density lipoprotein cholesterol and triglycerides and not in LDL cholesterol, which is also lower in people with lower CD4 lymphocyte counts [20,21].

We also wanted to observe whether the increase in the CD4 cell count and CD4:CD8 ratio after the introduction of suppressive ART may be associated with alterations in the lipid profile. In the analyzed group of patients, we did not observe a significant correlation between lipid profile changes and CD4 cell count recovery, but we observed that an increase in the CD4:CD8 ratio was associated with a decrease in triglyceride concentrations. However, that relationship was not statistically significant after adjustment for age, sex, chronic viral coinfections, and the scheme of ART.

The relationship between dyslipidemia and CD4 recovery is not fully understood. Interestingly, not only may CD4 influence lipid concentrations, but dyslipidemia may also play an important role in the pattern of immune recovery. A Chinese study suggested that higher baseline triglyceride concentrations could be associated with worse CD4 recovery [22]. Persistent inflammation in PWH may play an important role in the association between dyslipidemia and immune recovery. Both HIV infection and unfavorable lipid profiles are associated with systemic inflammation [23,24]. During ART, the majority of proinflammatory molecules decrease; however, they remain higher than those in healthy controls [25]. Persistent immune activation is an important cause of CD4 depletion and increases the serum levels of cholesterol and triglycerides in PWH [26,27]. Novel studies reporting the potential favorable impact of statins on inhibiting HIV infection and making CD4+ T cells less susceptible to HIV infection seem very promising in terms of further research on HIV infection, dyslipidemia, and CD4+ T-cell recovery [28].

In our study, we also assessed the impact of sex and chronic viral coinfections on the lipid profile. Although our study population included only 12 women, we observed significantly higher baseline total cholesterol, LDL cholesterol and non-HDL cholesterol concentrations in women than in men. It is believed that the risk of CVD in women with HIV is greater than that in men with HIV, which is the opposite of the general population [29]. Women with HIV are also considered more likely to have higher total, LDL and non-HDL cholesterol and higher triglyceride concentrations, which was also observed in our study [9]. However, no differences in lipid profiles were found among women and men after 1 year of antiretroviral treatment. We also did not observe any significant differences in lipid concentrations among individuals with HBV or HCV coinfection (baseline and after 1 year of ART); however, this may be due to the small number of patients with coinfections. Compared with HIV infection alone, HIV/HCV coinfection is generally associated with lower total cholesterol, LDL cholesterol, and HDL cholesterol, but cardiovascular risk seems to be greater among patients with coinfection, probably because of the greater prevalence of carotid plaques and endothelial dysfunction [10]. In contrast, HIV/HBV coinfection among patients receiving ART does not seem to be associated with time to CVD onset in comparison with HIV mono-infection [11].

The associations between lipid profiles and immune recovery during ART in PWH have not been explored. We believe that our study may contribute to its investigation; however, we acknowledge that it has several limitations. The population of our study group was small; therefore, the results may not apply to the whole population, especially in terms of sex or chronic viral coinfections. We also did not measure very low-density lipoprotein concentrations, which are often elevated in PWH, and observing their relationship with immune recovery could be valuable. Moreover, the observation period of 1 year was relatively short, which may not have allowed us to determine the long-term impact of dyslipidemia. However, we believe that this study will contribute to further research on the association between dyslipidemia and CD4 recovery.

Conclusion

Dyslipidemia is a prevalent problem in PWH, and the impact of immune recovery on the lipid profile is not well established. Healthcare providers should pay attention to the lipid profile in ART-naïve patients, especially those with a baseline CD4 cell count <200/μl. Lipid parameters should be precisely and regularly assessed in all PWH undergoing ART, since ART implementation may be associated with worsening of lipid parameters. A particular car is suggested for PWH with low baseline CD4 T-cell counts.

Declarations of competing interest

The authors have no competing interests to declare.

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Ethics approval

The study was conducted following the Declaration of Helsinki and approved by the Ethics Committee of the Medical University of Warsaw, Poland (AKBE/187/2023).

Author contributions

Conceptualization, methodology, software, data curation, formal analysis, writing—original draft preparation, visualization, project administration: AL. Validation, writing—review and editing: AZ, TM, and AW-D. Investigation: AZ and TM. Resources: AL, AZ, TM, and JK. Supervision: TM and AW-D. All authors have read and agreed to the published version of the manuscript.

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