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Lipid Profile and Cardiovascular Risk in HIV/AIDS Patients on Antiretroviral Therapy: Impact of Intervention on the Modifiable Risk Factors

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Authors' contributions

This work was carried out in collaboration between all authors. Author PRCA designed the study, wrote the protocol, collected the data, managed the analyses of the study, performed the statistical analysis, managed the literature searches and wrote the first draft of the manuscript. Author ECON wrote the protocol, managed the analyses of the study, performed the statistical analysis and wrote the first draft of the manuscript. Author FACP managed the literature searches and collected the data. Authors MDR and MFSMJ designed the study, managed the literature searches and collected the data. All authors read and approved the final manuscript.

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ABSTRACT

Aims: To evaluate the lipid profile and cardiovascular (CV) global risk of Brazilian HIV/AIDS patients before and after highly active antiretroviral therapy (HAART), and to study the impact of the intervention on the modifiable risk factors on CV risk.
Study Design: A prospective intervention study.
Place and Duration of Study: HIV/AIDS clinics of Teresópolis (RJ) - Brazil, between 2010 and 2012.

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Methodology: This study included 129 adult HIV-positive patients; HAART had been prescribed for a minimum period of one year. Patients were evaluated at 3 different stages: (1) Stage A: previously to HAART, (2) Stage B: after receiving HAART for 1 year, and (3) Stage C: 6 months after the intervention on the modifiable risk factors (treatment of dyslipidaemia, Diabetes Mellitus and arterial hypertension, encouragement of smoking cessation and institution of healthy dietary habits).
Results: A significant elevation of serum levels of cholesterol (186.1 ± 53.6 to 205.4 ± 49.5 mg/dL, P = .01) and a reduction of HDL-C (44.7 ± 6.3 to 42.3 ± 7.3 mg/dL, P = .02) was found after the introduction of HAART. Conversely, a decrease in total cholesterol levels was observed after the intervention on CV risk factors (205.4 ± 49.5 to 188.0 ± 62.8 mg/dL, P = .03). The CV risk was significantly reduced after the clinical intervention on modifiable risk factors (P = .02), based on the appropriate Brazilian guidelines.
Conclusion: The study is in accordance with the literature regarding the alterations of lipid profile and CV risk secondary to HAART. Despite its limitations, the present study supports the importance of screening and intervening on the modifiable risk factors to improve CV risk in the Brazilian HIV/AIDS population.

Keywords: HIV; AIDS; cardiovascular risk; HAART.

1. INTRODUCTION

The Acquired Immunodeficiency Syndrome (AIDS) represents one of the most important health issues of the present times due to its seriousness and its pandemic character.

In the beginning of the AIDS epidemics, most human immunodeficiency virus (HIV) carriers had a progressive deterioration of their immunologic functions with a fatal outcome. However, with the introduction of the highly active antiretroviral therapy (HAART), in 1996, an important change in the natural history of the disease, with a consequent impact on both its morbidity and mortality, was observed [1,2] With antiretroviral therapy treatment, patients were able to achieve undetectable viral loads with a progressive immunologic recovery and an acute drop in the rate of opportunistic infections and mortality [1].

With the increase in the survival rates after the introduction of HAART, there has been a significant modification of the disease spectrum: mortality directly related to HIV and opportunistic infections has been decreasing, while the incidence of cardiovascular disease (CVD) has been increasing [3-9].

Many studies have analyzed large databases and cohorts in the USA, Canada, and Europe in order to compare the incidence of CVD in HIV-positive patients. Despite the limitations (low number of events, short follow-up, incomplete evaluation of other risk factors), these studies have consistently reported a 1.5-fold increase in cardiovascular (CV) event rate in HIV-positive patients compared to controls [10-19].

In addition, CV risk adverse effects of HAART – such as lipodystrophy, disturbances in lipid and glucose metabolism - have become increasingly important in the morbidity and mortality of this population [20-26]. These alterations were initially attributed to the antiretroviral treatment with protease inhibitors (PI) [14,19,27,28]. Nevertheless, lipodystrophy was subsequently described in patients without PI treatment and it became clear that its origin is multifactorial, influenced by genetic factors, age, gender, period of exposure to antiretroviral therapy and to the HIV infection per se [29].

Although a number of studies have demonstrated a rise of CV risk during HAART [15,18,30] – even emphasizing that the incidence depends on the cumulative exposure to these drugs [27], the Strategies for Management of Antiretroviral Therapy (SMART) study, a large study that guided the interruption of HAART based on CD4 count, demonstrated that HAART disruption elevates CV risk even further. A possible explanation for such a finding is that the suppression of HIV may be cardioprotective through the reduction of cytokines [27,31,32].

Considering the above-mentioned data, HAART has modified the natural history of HIV infection, with a major impact on its survival outcome, resulting in an increase in the CV risk both due to the HIV infection per se and the antiretroviral

therapy. Since HAART interruption could lead to an increase of CV risk, viral load and a deterioration of the immune function, it is important to develop strategies that allow the identification of and the intervention on CV risk factors in the HIV-positive population.

The aim of the present study was to evaluate the lipid profile and CV global risk of Brazilian HIV/AIDS patients before and after HAART, and to study the impact of the intervention on the modifiable risk factors on CV risk.

2. METHODOLOGY

2.1 Participants

This prospective intervention study analysed a cohort of 129 adult patients with HIV/AIDS treated with antiretroviral drugs for at least one year.

2.2 Setting

The study was implemented at the HIV/AIDS clinics, which is part of the public health system of the city of Teresópolis (RJ) – Brazil.

2.3 Study Design

The authors initially invited 150 adult patients with HIV/AIDS that consecutively attended the HIV/AIDS clinics for the initiation of HAART to participate in the present study. Fourteen patients were lost during follow-up, four moved from Teresópolis and three decided to leave the study before it was finished.

The cohort was evaluated at three distinct stages: (1) Stage A: previously to HAART, (2) Stage B: after receiving HAART for 1 year, and (3) Stage C: 6 months after the intervention on the modifiable risk factors (treatment of dyslipidaemia, Diabetes Mellitus and arterial hypertension; encouragement of smoking cessation and implementation of healthy dietary habits).

By the time of the inclusion in the study (Stage A) and their routine consultations at the HIV/AIDS clinics of Teresópolis (RJ) – Brazil (Stages B and C), personal and clinical data (gender; age; HAART scheme; diagnosis and treatment of dyslipidaemia, arterial hypertension and Diabetes Mellitus; smoking and physical activity status) were obtained and physical examination (blood pressure, waist circumference, weight, and height) was performed. Subjects were submitted to laboratory exams (total cholesterol, HDL, triglycerides, fasting glucose), and the Framingham scale was applied in order to estimate global CV risk of patients [33].

Dyslipidaemia, arterial hypertension, and Diabetes Mellitus were treated according to the IV Brazilian Dyslipidaemia and Atherosclerosis Prevention, the VI Brazilian Hypertension, and the 2009 Brazilian Diabetes Society guidelines, respectively.

2.4 Definition of Terms

Dyslipidaemia was defined as the presence of at least one of the following: HDL bellow 40 mg/dL, in men, or 50 mg/dL in women; total cholesterol ≥ 240 mg/dL, and triglycerides ≥ 150 mg/dL [34].

Systolic arterial hypertension was defined as a systolic pressure above 130 mmHg and diastolic arterial hypertension as a diastolic pressure above 85 mmHg according to the criteria of the III report of the NCEP (National Cholesterol Education Program Criteria; 2002) [34].

Diabetes Mellitus was defined as the presence of fasting glucose levels > 125 mg/dL, on two or more occasions, or levels > 200 mg/dL after a 75g glucose load [35].

Obesity was defined as a BMI ≥ 30 kg/m² [36].

A waist circumference measure ≥ 94 cm, in men, or ≥ 80 cm, in women was considered high [37].

The diagnosis of Metabolic Syndrome was based on the presence of a high waist circumference and two or more of the following criteria: (1) fasting glucose > 100 mg/dL; (2) plasma triglycerides > 150 mg/dL; (3) plasma HDL < 50 mg/dL, in women, and < 40 mg/dL, in men; or (4) blood pressure > 130/85 mmHg or the treatment with antihypertensive drugs [37].

2.5 Intervention on Modifiable Cardiovascular Risk Factors

At the end of the routine consultations for the treatment of HIV/AIDS, two of the authors of the present study (P.R.C.A. and F.A.C.P.) took five minutes to educate the patients about the negative consequences of smoking, emphasizing the importance of smoking cessation with information about the options of adjunctive pharmacological treatment such as nicotine patches and bupropion.

Moderate physical activity initiation (or maintenance), moderate caloric restriction to induce a 5% weight loss in those who presented obesity and a change in dietary composition (for those with hypertension, diabetes and dyslipidaemia) were also emphasized [36]. Folders with dietary recommendations from the Brazilian Health Ministry were provided to the patients.

P.R.C.A. and F.A.C.P. also organized a discussion group on nutrition, smoking, and exercise that took place in the same days of the routine consultations.

The clinical intervention aimed at reducing the impact of these modifiable risk factors on CV risk to the lowest point on the Framingham score at Stage C.

2.6 Measures

Total body weight was measured on a standardized spring balance scale (Filizola, São Paulo, Brazil) with participants dressed uniquely in underwear. Weights were recorded to the nearest 0.1 kg.

Standing height was measured without shoes with a stadiometer (Filizola, São Paulo, Brazil) and recorded to the nearest 0.5 cm.

BMI was calculated by dividing total body weight (kg) to the squared standing height (m^2).

Waist circumference was measured with a non-elastic flexible measuring tape at the mid-distance between the lower rib and the iliac crest. Measures were recorded to the nearest 0.1 cm.

Tobacco load was calculated by multiplying the number of packs smoked daily by the number of years of smoking.

Blood pressure was measure in the right arm with a standard aneroid sphygmomanomater (Tycos, USA).

Reference values for laboratory exams were: Fasting glucose = 70-99 mg/dL, Total cholesterol < 200 mg/dL, HDL > 50 mg/dL (women) and > 40 mg/dL (men), Triglycerides < 151 mg/dL.

The Framingham cardiovascular score calculator was used to estimate the 10-year cardiovascular risk based on data such as gender, age, systolic blood pressure, treatment of hypertension, smoking, presence of diabetes mellitus, and levels of HDL and total cholesterol. The authors used the calculator available at [38]. The main outcome evaluated in the present study was the Framingham score of CV risk based on which patients were classified as having: (1) Low CV risk: absolute risk based on the Framingham score ≤ 10% in 10 years (excluding those with Diabetes Mellitus), (2) Medium risk: absolute risk based on the Framingham score between 10 and 20% in 10 years (excluding those with Diabetes Mellitus), (3) High risk: absolute risk based on the Framingham score ≥ 20%. Patients with Diabetes Mellitus were considered to have a high CV risk [39].

2.7 Statistical Analysis

Data are shown as mean ± SD, unless otherwise specified. Whenever quantitative analysis involving comparisons between means were necessary, the unpaired Student T test was used. In the case of the categorical variables, the Fisher's exact test of choice. When more than two groups were studied, categorical variables were analyzed using the chi-square test. Whenever necessary, data were transformed with the purpose of allowing the analysis by parametric tests. Measurements were log base 10 transformed (measurement = Log measurement), and ratios were square root transformed (ratio = square root of ratio). The Kolmogorov-Smirnov test was used to analyse the residuals for normality. (When alpha = 0.05, data passes this normality test). A $P < .05$ was considered statistically significant.

With a sample of 129 patients, the study had a 70% power to detect a difference between means of 17.57 with a significance level (alpha) of 0.05 (two-tailed).

The analyses were carried out using GraphPad Prism 6 for Windows (GraphPad Software, San Diego, California, USA), Epi Info™ 7 (Centers for Disease Control and Prevention, USA), and StatMate 2 for Windows Windows (GraphPad Software, San Diego, California, USA).

3. RESULTS AND DISCUSSION

3.1 Results

A total of 129 patients (women = 56.5%, male:female ratio = 0.7:1.3) with an age range from 20 to 74 years (mean 45 ± 10 years) were evaluated. Regarding antiretroviral therapy, the

most frequently used prescription scheme to treat these patients was an association of zidovudine, lamivudine, and efavirenz. In most patients, this scheme was maintained throughout the two years duration.

Table 1 exhibits the mean levels of lipids obtained in the present study and Table 2 presents the proportion of patients with lipid alterations. After HAART, there was a significant increase of total cholesterol (186.1 ± 53.6 to 205.4 ± 49.5 mg/dL, *P* = .01 – Table 1 and Fig. 1) and a significant reduction of HDL (44.7 ± 6.3 to 42.3 ± 7.3 mg/dL, *P* = .02 - Table 1 and Fig. 2). After the clinical intervention on CV risk factors, a decrease in total cholesterol levels was observed (205.4 ± 49.5 to 188.0 ± 62.8 mg/dL, *P* = .03 - Table 1 and Fig. 1). However, the distribution of patients with different degrees of increased cholesterol and triglycerides, as well as low HDL was not significantly different, when we compared the stages of the present study (Table 2).

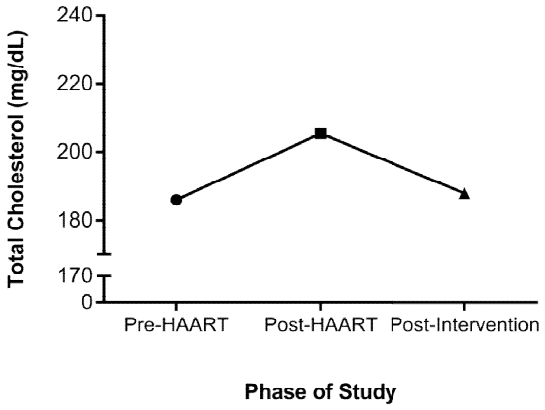

Fig. 1. Evolution of total cholesterol (Pre-HAART, Post-HAART, and Post-Intervention)

Table 3 provides comparisons of the prevalence of different CV risk factors between the three stages of the study, showing an increase of dyslipidaemia (from 31.0 to 69.8%; p < 0.01) and Diabetes Mellitus (from 1 to 4%; p = 0.01) after HAART, while the frequency of the other factors was not significantly modified after therapy. After the intervention on the potentially modifiable factors, the frequency of dyslipidaemia was significantly reduced to 58.1% (p = 0.03). In addition, the prevalence of tobacco smokers decreased from 29.6% (Post-HAART) to 27%, after intervention (p = 0.04). Finally, global CV risk is presented on Table 4 – there was a reduction on CV risk after the intervention measures (p = 0.02).

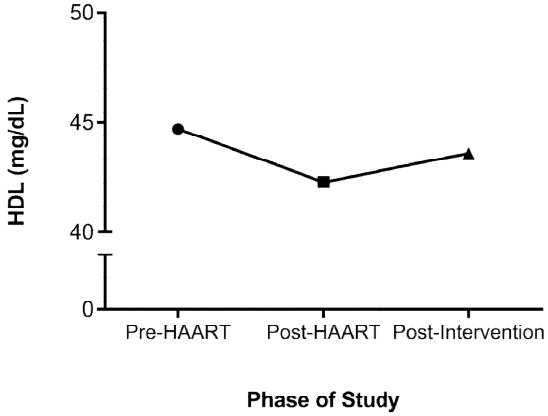

Fig. 2. Evolution of HDL (Pre-HAART, Post-HAART, and Post-Intervention)

3.2 Discussion

The predominance of females in the present study (1.3:0.7) is in accordance with a national trend (1.9:1) of feminization of the HIV/AIDS population [40]. This finding (56.5% of females), however, is in conflict with other studies, such as those by Friis-Moller et al. [27], Souza Neto et al. [41] and Arruda Jr et al. [42], in which the prevalence of women corresponded to 24.1%, 23.8%, and 23.8%, respectively.

Table 1. Lipids (Pre-HAART, Post-HAART, and Post-Intervention)

Lipids (mg/dL)	Pre-HAART	Post-HAART	DM Pre- & Post-HAART	*P*	Post-Intervention	DM Post-HAART & Intervention	*P*
Cholesterol	186.1±53.6	205.4±49.5	19.3	**.01**	188.0±62.8	-17.4	**.03**
HDL	44.7±6.3	42.3±7.3	-2.4	**.02**	43.6±7.6	1.3	.17
Triglycerides	186.8±112.8	193.5±120.0	6.7	.39	190.0±109.7	-3.5	.44

DM: Difference between means

Table 2. Distribution of dyslipidaemia (Pre-HAART, Post-HAART, and Post-Intervention)

Lipids (mg/dL)	Pre-HAART	Post-HAART	Post-Intervention
Cholesterol			
240-279	13.9%	14.7%	7.8%
≥ 280	4.7%	8.5%	9.3%
P	.43	.21	
HDL-c			
< 40	24.0%	34.1%	28.7%
≥ 40	76.0%	65.9%	71.3%
P	.10	.42	
Triglycerides			
150-199	11.6%	10.0%	10.0%
200-499	15.5%	17.0%	17.8%
≥ 500	3.9%	6.2%	4.6%
P	.81	.96	

Table 3. Prevalence of CV risk factors (Pre-HAART, Post-HAART, and Post-Intervention)

Risk factors	Pre-HAART	Post-HAART	*P*	Post-intervention	*P*
Dyslipidaemia	31.0%	69.8%	**< .001**	58.1%	**.03**
Diabetes Mellitus	1.0%	4.0%	**.01**	4.0%	**-**
Arterial hypertension	23.1%	24.4%	.15	24.4%	-
Smoking	29.6%	29.6%	-	27.0%	**.04**
Obesity	18.0%	19.1%	.15	18.0%	.15
Increased waist circumference	40.1%	41.8%	.14	41.8%	-
Metabolic Syndrome	18.3%	23.0%	.06	21.2%	.12

Table 4. Cardiovascular risk (Pre-HAART, Post-HAART, and Post-Intervention)

CV Risk	Pre-HAART	Post-HAART	Post-Intervention
Low	85.3%	81.4%	89.9%
Medium	10.0%	11.6%	6.2%
High	4.7%	7.0%	3.9%
P	.16	**.02**	

The mean age of 45 years in the present study approximates those reported by Friis-Moller et al. (39 years; IQR: 34-45) [27], Souza Neto et al. (43 ± 10.5 years) [41], and Arruda Jr et al. (39.5 ± 10 years) [42].

The high prevalence of some risk factors in our sample, is similar to findings of the Data collection on Adverse events of Anti-HIV Drugs (DAD) study that reported 45.9% of patients with dyslipidaemia, 8% with arterial hypertension, and 51.5% of smokers [27].

After HAART, this Brazilian cohort showed a statistically significant elevation of total cholesterol and reduction of HDL. The literature is unanimous regarding the demonstration of increased cholesterol with antiretroviral therapy, with possible concomitant elevation of triglycerides and decrease of HDL. It is important to emphasize that sometimes these alterations do not respond to pharmacological treatment of dyslipidaemia and a substitution of the antiretroviral medication may be necessary [41-47]. Taking this into consideration, in patients previously diagnosed with dyslipidaemia for whom antiretroviral therapy must be initiated, therapeutic schemes with low risk of adverse effects on lipid metabolism should be preferred [44,48,49].

In the present study, the prevalence of dyslipidaemia after the introduction of HAART was 68.9%, approximating that of the 67.5% reported by Souza Neto et al. [41], and higher than the 45.9%, by Friis-Moller et al. [27] and 36.6%, by Pupulin et al. [50].

Regarding the prevalence of hypercholesterolemia, the present study corroborates the data from two other Brazilian studies that reported indexes of 28% and 33%

[44,45] and the European DAD study that reported 21% prevalence [27]. Although our data indicate an increase in cholesterol levels following HAART, it is not in accordance with that of Diehl et al. [51]. In our study, treatment with atorvastatin was able to significantly reduce total cholesterol levels.

The prevalence of low HDL levels (24% pre-HAART and 34.1% post-HAART) is inferior to those obtained by Diehl et al., and Pupulin et al., and similar to that of Friis-Moller et al. that reported 68%, 83% and 26.1% prevalence, respectively [27,50,51].

The DAD study suggested that triglycerides levels are an independent CV risk factor for the HIV/AIDS population [27]. Hypertriglyceridemia was less frequent in the present sample than in one of the Brazilian studies used for comparison (55%) [51], higher than the in the other (21%) [50], but in accordance with the international data by Friis-Moller et al. (32.2%) [27]. In the present study, there was no difference between the three stages regarding the prevalence of hypertriglyceridemia. Nevertheless, the treatment protocol was limited by the unavailability of fibrates for the distribution by the Unified Health System – the public Brazilian Health system. Besides the non-pharmacological treatment with physical activity, fiber-rich diet, and low-saturated fat diet, pharmacological therapy with fish oil and niacin is recommended [44,52].

The first study to demonstrate a relationship between HAART and increased CV risk was the DAD – a prospective study involving 11 cohorts in 20 countries. It associated antiretroviral therapy with an increased risk of acute myocardial infarct during the first four to six years of treatment. The authors also indicated that risk factors such as age, smoking, gender, Diabetes Mellitus, and hypercholesterolemia were associated with hypertriglyceridemia and acute myocardial infarct [27].

A Slovenian study showed that 11.9% of HIV-positive patients on HAART presented a CV risk above 20% compared to only 5.3% of the control group. The main determinants for the increased CV risk in these patients were smoking, high total cholesterol, and low HDL levels. These authors also included patients who were initiating HAART by the time of the beginning of the study – this subgroup had a CV risk similar to the controls [53].

Regarding global CV risk evaluation according to the Framingham score: (1) 85.3% (Pre-HAART) and 81.4% (post-HAART) of our patients were low risk *versus* 71.6% found by Pupulin et al.; (2) 10% (Pre-HAART) and 11.6% (Post-HAART) had intermediate risk, contrasting with 25% obtained by Pupulin et al.; (3) 4.7% (Pre-HAART) and 7% (Post-HAART) had high risk *versus* 3.4% in the study of Pupulin et al. [3]. In the present study, 14.7% (Pre-HAART) and 18.6% (Post-HAART) presented a CV risk above 10% in 10 years, contrasting with data from Leite et al. in which 53% had a risk above 10% in 10 years [54].

In 2013, Souza-Neto et al. [41] estimated the impact of the hypothetical treatment of CV risk factors of HIV/AIDS patients and concluded that it is possible to decrease the impact of HAART on the global CV risk. Our data support their findings, since a reduction of CV risk was obtained after the intervention on the modifiable risk factors. A drop in CV related morbidity and mortality due to this strategy is expected in these patients. The DAD study showed a reduction of CV related deaths from 1.8 to 0.9 person-years [55].

Despite the rising importance of CV morbidity and mortality for HIV-infected patients, the strategies to minimize CV disease are not always implemented. An example is a study among 397 HIV-positive individuals that fulfilled the criteria for the utilization of aspirin for primary prevention of CVD based on the Framingham score – medication was prescribed for only 17% of the study participants [56]. These numbers call our attention to the importance of educating the health professionals on the strategies for CV risk reduction for HIV-positive subjects.

The best approach in order to reduce CV risk in HIV-positive patients has not been precisely defined, but it is largely accepted that the same techniques used in non-HIV patients may be applied [48,57,58]. Patients with dyslipidaemia, glucose abnormalities or arterial hypertension should maintain a healthy diet, physical activity, and quit smoking [48]. The choice of antiretroviral drug should be individualized, and it is important to use a multi-professional approach to help these patients identify and modify the CV risk factors.

Physical exercise and weight loss should be emphasized in any CV event prevention program, especially in the HIV-positive patients

on HAART, due to the high prevalence of dyslipidaemia and lipodystrophy [59-63]. The benefit of diet orientation is supported by a study that included 83 HIV-positive patients that initially received dietary orientation by the time of the beginning of HAART and then were randomized for trimestral dietary consultations or no dietary follow-up. The study concluded that the intervention group had a decrease of triglycerides, while the control showed increases of total cholesterol, LDL, triglycerides, and BMI [60]. Other studies have also demonstrated improvement in dyslipidaemia, waist circumference, blood pressure control, and HbA1c with dietary and exercise programs [61-63].

The indications for lipid-lowering therapy in primary prevention of CVD for HIV-positive individuals are the same as those for non-HIV subjects. Statin therapy reduces relative CV risk in 20 to 30%. Consequently, the absolute benefit of statin therapy is proportional to the individual's CV risk [49]. Since statins may have important pharmacological interactions with the antiretroviral drugs, the studies suggest atorvastatin, rosuvastatin, and pitavastatin as viable options [49,64-66]. However, for those treated with ritonavir, pitavastatin is particularly indicated, due to less potential for interaction. For the non-ritonavir patients, atorvastatin is the drug of choice because of its efficacy and broader clinical experience [43]. Pravastatin is an acceptable alternative, since it is not metabolized by CYP3A4; although, not as efficient as the other three in the reduction of LDL [66,67]. The prescription of simvastatin should be avoided in HIV-infected patients on HAART due to the potential to serious adverse reactions, such as rhabdomyolysis [44,49].

Treatment of hypertriglyceridemia in HIV-positive subjects follows the same rationale as in the general population. Fibrates may be used previously to statins in patients with triglycerides levels above 500 mg/dL [44]. In opposition to the statins, these agents are metabolized by CYP4A and are not susceptible to significant pharmacological interactions with antiretroviral drugs. In patients in need of additional triglyceride-lowering therapy, the inclusion of fish oil or niacin may be useful [68-71].

Fibrates may be combined with statins in patients with high CV risk. Nevertheless, these patients should be closed monitored for the development of myositis [62,71].

The present study was limited by: (1) the impossibility of prescription of fibrates, fish oil, and niacin for the treatment of hypertriglyceridemia, since these drugs are not provided by the Brazilian public health system; (2) the absence of participation of nutritionists and physical trainers in the attending team during the three stages of the study; and (3) the impossibility of prescription of pharmacological support for those patients who tried to abandon tobacco smoking during the study, since these drugs are not provided by the Brazilian treatment program for HIV/AIDS.

The authors would also like to acknowledge the limited power to detect differences between the post-HAART and the intervention stages and the absence of a control group. Therefore, despite the reduction of CV risk after six months of intervention, our results should be corroborated by more robust studies to the point in which the importance of the intervention on modifiable risk factors can be accurately investigated and influence clinical decisions during the treatment of HIV/AIDS patients.

4. CONCLUSION

The present study revealed a significant increase in total cholesterol and reduction of HDL after HAART. It also identified an elevation in the CV risk after a period of 12 months of antiretroviral drug treatment and a reduction of the prevalence of dyslipidaemia and smoking, with a significant decline in the CV risk after the intervention on the modifiable risk factors, based on the appropriate Brazilian guidelines.

It is in accordance with the literature regarding the alterations of lipid profile and CV risk secondary to HAART. Despite its limitations, the present study supports the importance of screening and intervening on modifiable risk factors to improve CV risk in the Brazilian HIV/AIDS population.

INFORMED CONSENT AND ETHICAL APPROVAL

The present study was approved by the research and ethics committee of the Serra dos Órgãos University Center and was therefore performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. Informed consent was obtained from all the patients. All the individual patient data were accessed solely with academic purposes.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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