Serum lipid and glucose profiles in HIV-positive Nigerian children

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Abstract

Objectives: To describe the fasting serum lipid and glucose profiles of HIV-positive Nigerian children and determine the prevalence and risk factors for dyslipidaemia and hyperglycaemia, which are risk factors for cardiovascular diseases.

Methods: This was a comparative cross-sectional study carried out at the Paediatric Infectious Disease Clinic (PIDC) of the Jos University Teaching Hospital (JUTH) for HIV-positive children and at two primary schools in Jos for HIV-negative children as controls. One hundred and forty-two HIV-positive children aged 6–18 years and an equal number of controls were studied by determining their fasting serum lipid and glucose levels. The prevalence of dyslipidaemia and hyperglycaemia was determined and their risk factors obtained using multivariate logistic regression. *P* values of less than 0.05 were considered statistically significant.

Results: Mean triglyceride levels were significantly higher in HIV-positive children compared with controls at 87.2 mg/dL (95% confidence interval [CI] 79.4–95.0) and 68.1 mg/dL (95% CI 62.5–72.7), respectively (*P*<0.001). There were no significant differences in mean glucose levels. Dyslipidaemia was significantly higher in HIV-positive children (21.8%) compared with controls (12.7%; *P*=0.04). Total serum cholesterol was elevated in 17 (12.0%) HIV-positive participants compared with seven (4.9%) of controls (*P*=0.02). Children on lopinavir/ritonavir (LPV/r) and those with no significant or mild disease had a significantly higher prevalence of hypercholesterolaemia (33.3% *vs* 4.8% and 14.5% *vs* 0.0%, respectively; *P*<0.001).

Conclusion: HIV-positive children on antiretroviral (ARV) drugs, especially LPV/r, should have their lipids regularly monitored as those with dyslipidaemia stand the risk of subsequently developing cardiovascular diseases.

Keywords: dyslipidaemia, hyperglycaemia, cardiovascular disease risk, HIV-positive children, Nigeria

Introduction

With approximately 35 million individuals being infected with HIV/AIDS worldwide, the dangers of this disease cannot be overemphasised [1]. Two-thirds of these individuals live in sub-Saharan Africa and in 2014, an estimated 3.4 million individuals were living with HIV/AIDS in Nigeria, approximately 10% of whom were children [2].

The improvement in the survival rates for both adults and children living with HIV resulting from the use of antiretroviral (ARV) drugs has brought with it the metabolic effects of some of these drugs including hyperglycaemia and dyslipidaemia including increased total cholesterol (hypercholesterolaemia), increased low density lipoprotein cholesterol (LDL-c), decreased high density lipoprotein cholesterol (HDL-c) and hypertriglyceridaemia. These effects are established risk factors for cardiovascular diseases (CVDs) such as coronary artery disease, myocardial infarction, angina and stroke as they promote plaque formation, leading to arterial narrowing, which can contribute to significant morbidity and mortality later in life [3,4]. In addition to the effects of ARVs, HIV itself can also directly induce endothelial cell injury resulting in arterial plaque formation [5]. HIV-positive children may have a higher risk of developing CVD later in life than HIV-negative children as a result of these metabolic complications.

Dyslipidaemia and hyperglycaemia have been shown to be more prevalent in individuals with HIV/AIDS, especially those on ARVs, compared with the general population [6]. All classes of ARV have been shown to be associated with metabolic effects but protease inhibitors (PIs) are particularly associated with the development of hyperlipidaemia because they inhibit differentiation of

adipocytes and increase lipolysis leading to hypercholesterolaemia, hypertriglyceridaemia and an increase in LDL cholesterol [7]. PIs also reduce glucose transportation inducing insulin resistance (IR), which will lead to hyperglycaemia [7,8]. The nucleoside inhibitors of retrotranscriptase (NRTIs), especially stavudine (d4T), also inhibit mitochondrial DNA polymerase within adipocytes that results in mitochondrial injury, and this leads to hypercholesterolaemia and hypertriglyceridaemia [8].

The metabolic effects of HIV and/or ARV drugs in HIV-positive children in Jos, Nigeria has, however, not been studied and currently data on the lipid and glucose profile in the Nigerian paediatric HIV population are sparse [9]. Closing the knowledge gap will help in adequate screening and risk stratification of these children in order to prevent later development of CVD [10]. The present study, therefore, aims to describe the prevalence and risk factors for dyslipidaemia and hyperglycaemia in HIV-positive Nigerian children compared with age- and sex-matched controls. This would help to improve the level of care in these children and open opportunities for further longitudinal research.

Methods

Study population and site

Children aged 6–18 years of age and on follow-up at the Paediatric Infectious Disease Clinic (PIDC) of the AIDS Prevention Initiative in Nigeria/President's Emergency Program For AIDS Relief (APIN/PEPFAR) of the Jos University Teaching Hospital in Nigeria were recruited into the study. The PIDC serves children, infected or exposed to HIV/AIDS who live in Jos, the capital of Plateau State, its environs and also neighbouring states in the north-central part of Nigeria.

One hundred and forty-two HIV-positive children were selected sequentially as they presented to the clinic. An equal number

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of, presumably healthy, HIV-negative age- and sex-matched controls from primary/secondary schools were recruited for the study.

Inclusion criteria:

■ HIV-positive children, 6–18 years as participants

■ HIV-negative children, 6–18 years as controls

■ Children whose primary caregiver had given consent/assent

Exclusion criteria:

■ Children <6 years of age

■ Members of the control group who tested positive for HIV infection

■ Members of the control group with any known chronic medical illness

Biodata and anthropometry

A history of first- or second-hand smoking was obtained. First-hand smoking is defined as smoke inhaled directly by the smoker while second-hand smoking is defined as a combination of smoke from the burning end of a cigarette and the smoke breathed out by smokers who live with children [11]. A known family history of CVD including hypertension, sudden heart attack and stroke was also obtained from the primary caregiver or by phone call to a first-degree relative [12]. The socio-economic status of the children was obtained using the Olusanya classification [13].

Weight and height were measured using standard methods and the Quetelet index was used to calculate body mass index (BMI) [14]. Further data obtained on the HIV-positive participants included CD4 cell count, viral load, World Health Organization (WHO)-defined clinical and immunological stages of the disease and ARV drugs used by the children [15]. All data obtained were recorded on a predesigned questionnaire.

Laboratory investigations

A serum lipid profile that included total cholesterol, HDL-cholesterol (HDL-c), LDL-cholesterol (LDL-c) and triglycerides (TG), and glucose was analysed from a fasting blood sample using the enzymatic colorimetric assay and hexokinase G6PDH/UV method, respectively. The equipment used for the analysis was the Roche Cobas 311 manufactured in 2002.

Hypercholesterolaemia was defined as a total cholesterol level ≥200 mg/dL and a serum triglyceride level ≥150 mg/dL was considered as hypertriglyceridaemia. Increased LDL-c and decreased HDL-c were defined as levels ≥130 mg/dL and <40 mg/dL, respectively [16]. Dyslipidaemia was said to be present if the child had any of the lipid abnormalities. Hyperglycaemia was defined as a fasting blood sugar (FBS) >110mg/dL [16].

For HIV-positive children, a CD4 cell count performed within a 3 month period from the time of study and the worst ever clinical stage of disease were obtained from the case records of each participant and documented. Using WHO criteria, CD4 cell counts for each child were used to characterise current immunological stage into 'not significant' (>500 cells/mm³), 'mild' (350–499 cells/mm³), 'advanced' (201–349 cells/mm³) or 'severe' <200 cells/mm³ immunodeficiency [15]. Blood samples were taken from the control group to assess their HIV status using the rapid diagnostic test after pre-test counselling. Two children were found to be positive and sent to a tertiary hospital for further testing and evaluation.

Ethical considerations

Ethical clearance was obtained from the Jos University Teaching Hospital Ethical Review Board. Written and informed consent was obtained from the parents and, subsequently, assent from all the children. Consent was obtained from the parents of members of the control group to perform HIV testing on their children.

Statistical analysis

Data were entered into the Epi Info 7.0 statistical software. Frequency tables were used to present qualitative data while quantitative data was presented using the mean and 95% confidence interval (CI). A chi-squared statistical test was used to determine the relationship between dyslipidaemia and certain risk factors. Bivariate logistic regression was used to initially identify variables associated with each outcome: variables that were significantly associated with the outcome were then fitted into a multivariate logistic regression model to determine the risk factors for dyslipidaemia. Odds ratio (OR) with their CI were obtained and *P* values ≤0.05 were considered statistically significant.

Results

Characteristics of study population

The two groups comprising 142 HIV-positive children (69 males, 48.6%; 73 females, 51.4%) and the same number of HIV-negative children both had a male to female ratio of 1.1:1 and a mean age at enrolment of 10.6 years (95% CI 10.1–11.1) and 10.8 years (95% CI 10.3–11.3), respectively (*P*=0.64). HIV-positive children were from a significantly lower socio-economic group compared with the controls (*P*<0.001). The prevalence of second-hand smoking was significantly higher in HIV-positive children (16.9% *vs* 2.8%, respectively, *P*<0.001) but there was no difference in the presence of a known family history of CVD in the two groups (*P*=0.53) (Table 1).

A total of 140 (98.6%) HIV-positive children were on ARV drugs while two (1.4%) were not. Of the 140 on ARV drugs, 104 (72.6%) were on first-line drug therapy with a combination of two NRTIs and one non-nucleoside reverse transcriptase inhibitor (NNRTI) while 36 (25.7%) participants were on second-line drugs – two NRTIs and one PI, lopinavir/ ritonavir (LPV/r). The various combinations of ARV drugs received by the participants are shown in Table 1.

The HIV-positive participants had a mean age at diagnosis of 4.9 years (95% CI 4.2–5.6) and a mean current CD4 cell count of 698.4 cells/mm³ (95% CI 633.6–763.2). Sixty-four (45.1%) participants had a WHO clinical stage of 1, while 26 (18.3%), 46 (32.4%) and 6 (4.2%) had clinical stage 2, 3 and 4, respectively. For the current WHO immunological stage of disease, 97 (68.3%) children had no significant immunodeficiency, while 20 (14.1%) had mild disease and 11 (7.7%) and 14 (9.9%) had advanced and severe disease, respectively. All HIV-positive children were likely to have been infected perinatally as their biolgical mothers were HIV positive.

Mean clinical and laboratory parameters in HIV-positive children and controls

The HIV-positive children were significantly lighter and shorter compared with the control group, but their BMI did not differ significantly. The control group had a higher mean FBS level compared with the HIV-positive children (*P*=0.009), while the latter had a significantly higher mean serum triglyceride levels (*P*<0.001). The mean total cholesterol levels were similar in both groups (*P*=1.0). Although the control group had a higher mean LDL cholesterol and a lower mean HDL cholesterol, the difference was not statistically significant (Table 2).

Table 1. Socio-demographic characteristics of 142 HIV-positive children and age and sex-matched HIV-negative controls

Characteristics	Total participants =284 n (%)	HIV-positive participants =142 n (%)	HIV-negative participants =142 n (%)	P
Mean age in years (95%CI)	10.7 (10.3–11.1)	10.6 (10.1–11.1)	10.8 (10.3–11.3)	0.64
Sex				
Male	138 (48.6)	69 (48.6)	69 (48.6)	
Female	146 (51.4)	73 (51.4)	73 (51.4)	1.0
Socio-economic status				
Upper	109 (39.2)	24 (16.9)	85 (61.2)	
Middle	79 (28.4)	36 (25.4)	43 (30.9)	
Lower	90 (32.4)	82 (57.8)	11 (7.9)	<0.001
Second-hand smoking				
Yes	28 (9.9)	24 (16.9)	4 (2.8)	
No	256 (90.1)	118 (83.1)	138 (97.2)	<0.001
Known family history of heart disease				
Yes	97 (34.2)	51 (35.9)	46 (32.4)	
No	183 (64.4)	87 (61.3)	96 (67.6)	
Unknown	4 (1.4)	4 (2.8)	4 (2.8)	0.53
ARV therapy		140 (98.6)		
First-line drugs		104 (72.3)		
NVP+3TC+ZDV		64 (45.1)		
ZDV+3TC+EFV		34 (23.9)		
3TC+EFV+ABC		4 (2.8)		
NVP+3TC+d4T		1 (0.7)		
NVP+3TC+ABC		1 (0.7)		
Second-line drugs				
3TC+ABC+LPV/r		36 (25.4)		

ARV: antiretroviral; ABC: abacavir; d4T: stavudine; EFV: efavirenz; LPV/r: lopinavir/ritonavir; NVP: nevirapine; ZDV: zidovudine; 3TC: lamivudine

Table 2. Comparison of clinical and laboratory parameters

Parameters	All participants Mean (95%CI)	HIV-positive Mean (95%CI)	HIV-negative Mean (95%CI)	P
Weight (Kg)	34.6 (33.1–6.1)	31.9 (30.1–33.7)	37.2 (34.9–39.5)	<0.001*
Height (cm)	137.1 (135.2–139.0)	132.0 (129.4–134.6)	142.1 (139.5–144.7)	<0.001*
BMI (Kg/m²)	17.8 (17.5–18.1)	17.8 (17.4–18.2)	17.8 (17.2–18.4)	0.8
FBS (mmol/L)	79.5 (78.4–80.6)	78.0 (76.4–79.6)	81.0 (79.4–82.6)	0.009*
Total cholesterol (mg/dL)	156.6 (152.3–160.9)	156.6 (149.4–163.8)	156.6 (151.7–161.5)	1.0
HDL-c (mg/dL)	61.0 (58.9–63.1)	62.9 (59.7–66.1)	59.0 (56.3–61.7	0.07
LDL-c (mg/dL)	81.2 (77.2–85.1)	77.4 (70.6–84.1)	85.0 (80.9–89.0)	0.06
Triglycerides(mg/dL)	77.7 (72.8–82.6)	87.2 (79.4–95.0)	68.1 (62.5–72.7)	<0.001

* Statistically significant
BMI: body mass index; FBS: fasting blood sugar; HDL-c: high density lipoprotein cholesterol; LDL-c: low density lipoprotein cholesterol

Prevalence of dyslipidaemia and hyperglycaemia in HIV-positive children and controls

Dyslipidaemia was present in 21.8% and 12.7% of the HIV-positive children and the control group, respectively, with the difference being statistically significant (P=0.04). Of the 142 HIV-positive children, 17 (12.0%) had hypercholesterolaemia compared with 7 (4.9%) in the control group (P=0.03). There was no difference in the prevalence of hypertriglyceridaemia, decreased HDL-c and increased LDL-c in both groups (Table 3). The two HIV-positive children who were not on ARV drugs had no dyslipidaemia.

Factors associated with dyslipidaemia in HIV-positive children and controls

Among HIV-positive children, those who received an ARV drug combination therapy with LPV/r were significantly more likely to have hypercholesterolaemia, hypertriglyceridaemia and increased LDL-c (Table 4). Furthermore, children with a longer duration on LPV/r were more likely to have hypercholesterolaemia: 221.8 weeks (95% CI 209.8–233.8) *vs* 141.3 weeks (95% CI 123.6–159.0; P=0.02). The duration on LPV/r did not, however, affect the prevalence of hypertriglyceridaemia: 190.5 weeks (95% CI 174.2–206.8) *vs* 163.1 weeks (95% CI 145.7–180.5 weeks;

P=0.66); or high LDL-c: 185.3 weeks (95% CI 169.3–201.3) *vs* 163.7 weeks (95% CI 146.1–181.3; *P*=0.59). Children with no significant and mild disease had a significantly higher prevalence of hypercholesterolaemia (*P*=0.01). When multivariate logistic regression was used, there was still a significant association between the use of LPV/r, immunological staging and the presence of hypercholesterolaemia.

The finding of decreased HDL-c in the HIV-positive children was higher in females (Table 5). There was no association among any of the dyslipidaemias, the age at diagnosis and WHO clinical stage of disease in the children. There was no association among age, socio-economic status, second-hand smoking, known family history of CVD and dyslipidaemia in both HIV-positive children and controls.

Discussion

This descriptive cross-sectional study investigated serum lipid and glucose profiles, which are documented risk factors for later development of CVD, in 142 HIV-positive children and compared them with age- and sex-matched controls. It also identified the relationship of certain parameters such as age, clinical and immunological stage of disease, and the use of various ARV combinations between dyslipidaemia in participants.

The prevalence of dyslipidaemia in HIV-positive children in this study was 21.8%, a finding that is similar to that from other studies, ranging between 19.3% and 48% [17–19]. In this study, hypercholesterolaemia was the most common type of dyslipidaemia

Table 3. Prevalence of dyslipidaemia and hyperglycaemia in HIV-positive participants age- and sex-matched HIV-negative controls

Abnormality	Total participants =284 n (%)	HIV-positive participants =142 n (%)	HIV-negative participants =142 n (%)	P
Dyslipidaemia	49 (17.3)	31 (21.8)	18 (12.7)	0.04*
Hypercholesterolaemia	24 (8.5)	17 (12.0)	7 (4.9)	0.03*
Low HDL-c	13 (4.6)	7 (4.9)	6 (4.2)	0.78
High LDL-c	15 (5.3)	9 (6.3)	6 (4.2)	0.43
Hypertriglyceridaemia	18 (6.3)	13 (9.2)	5 (3.5)	0.05
Hyperglycaemia	2 (0.7)	1 (0.7)	1 (0.7)	1.00

* Statistically significant
HDL-c: high density lipoprotein cholesterol; LDL-c: low density lipoprotein cholesterol

Table 4. Factors associated with hypercholesterolaemia and hypertriglyceridaemia in the HIV-positive participants

Characteristic (*n*)	Raised cholesterol, total participants=17		P	Raised triglycerides, total participants=13		P
	n (%)	OR (95% CI)		n (%)	OR (95%CI)	
Sex						
Male (69)	10 (14.5)			7 (10.1)		
Female (73)	7 (9.6)	1.6 (0.6–4.5)	0.19	6 (8.2)	1.3 (0.4–4.0)	0.35
Socio-economic status						
Upper (24)	1 (4.2)			2 (8.3)		
Middle (36)	8 (22.2)	6.6 (0.8–56.5)	0.09	3 (8.3)	1.0 (0.2–6.5)	1.0
Lower (82)	8 (9.8)	2.5 (0.3–20.9)	0.40	8 (9.8)	1.2 (0.2–6.0)	0.8
Second-hand smoking						
Yes (24)	1 (5.8)			1 (4.2)		
No (118)	16 (94.2)	0.3 (0.01–1.7)	0.36	12 (10.2)	0.4 (0.01–2.4)	0.7
Known family history of heart disease						
Yes (51)	6 (11.8)			3 (5.9)		
No (87)	10 (11.5)	1.2 (0.4–2.5)	0.98	10 (11.5)	1.6 (0.6–4.4)	0.92
BMI						
Normal (125)	16 (12.8)			12 (9.6)		
Low (3)	0 (0.0)			1 (7.7)		
High (14)	1 (7.1)	0.5 (0.06–4.3)	0.5	0 (0.0)	4.7 (0.4–55.8)	0.2
WHO clinical staging						
1 and 2 (90)	11 (12.1)			8 (8.9)		
3 and 4 (52)	6 (11.8)	1.0 (0.3–2.8)	0.9	5 (9.6)	1.1 (0.3–3.5)	0.88
WHO immunological staging						
Not significant and mild (117)	17 (14.5)			9 (7.7)		
Advanced and severe (25)	0 (0.0)	**	0.01	4 (16.0)	2.3 (0.6–8.0)	0.11
ARV combinations						
First-line (104)	5 (4.8)			6 (5.8)		
Second-line (36)	12 (33.3)	9.9 (3.2–30.8)	<0.001	7 (19.4)	3.9 (1.2–12.7)	0.01

** Undefined
ARV: antiretroviral; BMI: body mass index; WHO: World Health Organization

Table 5. Factors associated with low HDL-c and high LDL-c in the HIV-positive participants

Characteristic (n)	Lowered HDL-c, total participants=7		P	Raised LDL-c, total participants=9		P
	n (%)	OR (95% CI)		n (%)	OR (95%CI)	
Sex						
Male (69)	1 (1.4)			7 (10.1)		
Female (73)	6 (8.2)	1.6 (0.6–4.5)	0.19	6 (8.2)	1.3 (0.4–4.0)	0.35
Socio-economic status						
Upper (24)	1 (4.2)			1 (4.2)		
Middle (36)	3 (8.3)	2.1 (0.2–21.4)	0.54	4 (11.1)	2.9 (0.3–27.4)	0.36
Lower (82)	3 (3.7)	0.8 (0.09–8.8)	0.91	4 (4.9)	1.2 (0.1–11.1)	0.89
Second-hand smoking						
Yes (24)	3 (12.5)			1 (4.2)		
No (118)	4 (3.5)	4.0 (0.7–20.8)	0.06	8 (6.8)	0.6 (0.03–4.0)	0.35
Known family history of heart disease						
Yes (51)	3 (5.9)			3 (5.9)		
No (87)	4 (4.6)	1.2 (0.4–3.7)	0.77	6 (6.9)	0.7 (0.2–2.2)	0.51
BMI						
Normal (125)	5 (4.0)			12 (9.6)		
Low (3)	1 (33.3)	12 (0.9–155.5)	0.06			
High (14)	1 (7.1)	1.8 (0.2–17.0)	0.59	1 (7.1)	1.1 (0.1–9.7)	0.92
WHO clinical staging						
1 and 2 (90)	6 (6.7)			4 (3.4)		
3 and 4 (52)	1 (14.3)	0.3 (0.01–1.9)	0.11	3 (12.0)	3.8 (0.7–9.7)	0.06
WHO immunological staging						
Not significant and mild (117)	4 (3.4)			8 (6.8)		
Advanced and severe (25)	3 (12.0)	3.9 (0.8–18.4)	0.06	1 (4.0)	0.6 (0.02–3.8)	0.34
ARV combinations						
First-line (104)	5 (4.7)			1 (0.9)		
Second-line (36)	2 (5.9)	1.3 (0.2–6.7)	0.42	8 (23.5)	31.3 (4.7–728.1)	<0.001*

* Statistically significant
ARV: antiretroviral; BMI: body mass index; HDL-c: high density lipoprotein cholesterol; LDL-c: low density lipoprotein cholesterol; WHO: World Health Organization

found and was present in 12.2% of the HIV-positive participants. This is similar to the 13% reported in the US and 14.8% in India [18], but lower than the 27.2% detected by a study in Spain [20].

In studies where more HIV-positive children have advanced disease, hypertriglyceridaemia is a common finding. Blazquez et al. reported a prevalence rate of 39.8% among HIV-positive children in Spain where 67% of the participants had severe disease and a rate of 83% in a study in Uganda where up to 65% of the children had severe HIV infection. In this study, only 10% of participants had severe infection, which could explain the lower prevalence rate of hypertriglyceridaemia (8.6%) obtained [20,21].

Protease inhibitors (PIs) have been significantly associated with dyslipidaemia in other studies [22], a finding also in this study where the use of second-line ARV combinations that contained the protease inhibitor LPV/r was significantly associated with hypercholesterolaemia, hypertriglyceridaemia and increased LDL-c when compared with participants on first-line drugs. In a study by Aldrovandi et al. [23], PI use was associated with elevated triglycerides among 52% of HIV-positive children while Carter et al. found a significant association between the use of multiple PIs and hypercholesterolaemia [24]. Kamara et al. found that individuals on LPV/r had the worst triglyceride profile compared with those on other PIs [25].

In Aldrovandi et al. [23] the use of the NNRTIs nevirapine (NVP) and efavirenz (EFV) was linked to a high HDL-c value, also known

as 'good' cholesterol. Therefore, it is not surprising that 95.1% of the HIV-positive children in this study had high HDL-c levels because 72% of them were on ARV combinations that contained either NVP or EFV. A study in Zimbabwe found that children on (zidovudine) ZDV had lower total cholesterol, HDL-c and TG than those on NVP or EFV [26]. Also, decreased serum concentrations of HDL-c in individuals who are ARV naive has been shown to point to chronic inflammation [27]. Participants in this study had a significantly higher mean serum HDL-c compared with the control group, which could imply good control of chronic inflammation. Decreased HDL-c was the least common of the dyslipidaemias and this has also been shown in several other studies [28–30].

Only one study participant and one control group member had an FBS level of greater than 100mg/dL, a finding that is in keeping with studies from Brazil and Thailand where no children had elevated FBS levels [31,32]. The latter study showed that the prevalence of increased FBS was low in patients on NNRTIs (NVP or EFV), a finding also present in this study. The child who had elevated FBS was, however, on an ARV combination that had two NRTIs (lamivudine and abacavir) and efavirenz, a combination that has been shown to be associated with insulin resistance [33]. There were only three children on this ARV combination and this number is too small to draw up any statistically meaningful conclusions.

The virus itself could initiate a systemic inflammatory response in an individual as a result of persistent infection leading to hypertriglyceridaemia [34]. In this study, only two children who were newly diagnosed with HIV, were not yet on ARV drugs and had no lipid abnormalities. However, this number is too small to make any statistical conclusion.

The present study shows that the mean weight and height were significantly lower in the HIV-positive participants when compared with the control group, a finding that has been found in several studies. It has been attributed to the chronic nature of the disease with its related increase in metabolism and caloric demands, comorbidities, medications and food insecurity [35]. In the study population, a higher proportion had a lower socio-economic background, which raises the question of the availability of adequate nutrition [36].

An inverse relationship has been found between socio-economic status and smoking, a finding also observed in this study where second-hand smoking was more commonly found in the participants from a lower socio-economic background [37,38].

Limitations

Due to cost implications, we were not able to assess the presence and prevalence of insulin resistance. There were also very few HIV-positive children attending the PIDC who were not on ARVs making it difficult to compare the effects of ARVs as a whole on lipid profile in HIV-positive children.

Conclusion

Regular monitoring of the lipid profile of HIV-positive children, especially those on LPV/r, will be clinically useful in determining those who may have a higher CVD risk. Longitudinal and larger multicentre studies will be useful in determining the progression of dyslipidaemias in HIV-positive children over time. Because the safety of only a few ARV drugs has been well established in children, more research is needed to broaden the choice of drugs available for treating HIV infection in these individuals where it may be necessary to switch to other types of PI-based therapy or a different class of ARV [39,40].

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Declaration of interests

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