Original Article

Electrocardiographic Abnormalities in Children with Human Immunodeficiency Virus Infection Presenting to the Federal Medical Centre, Umuahia, South-east Nigeria

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Abstract

Background: With the availability of highly active anti-retroviral therapy and attendant increased lifespan of human immunodeficiency virus (HIV)-infected children, late complications of the disease, especially cardiovascular complications have become a growing problem for them. The cardiovascular complications of HIV infection start early in the course of the disease, although may remain asymptomatic until later in life, when they manifest with life-threatening symptoms. The electrocardiogram (ECG) is an invaluable tool in the early diagnosis of these abnormalities. **Aim:** This study assessed the prevalence and types of ECG abnormalities among HIV-infected children. **Patients, Materials and Methods:** It was a hospital-based, comparative, cross-sectional study involving randomly selected HIV-infected children (subjects) and age- and gender-matched HIV-uninfected children (controls). Relevant information was obtained through questionnaires, medical records, and physical examination. All participants underwent a 12-lead ECG assessment. **Results:** Electrocardiographic abnormalities were observed in 42.9% of subjects compared to 17.9% of controls (χ^2 = 8.28; P = 0.004). The participants were about three times more likely to have ECG abnormalities than controls (odds ratio = 3.45, 95% confidence interval = 1.45–8.19). Left ventricular hypertrophy (LVH) was the most common abnormality in the subjects (14.3%), and compared to controls (1.8%), this was significant (χ^2 = 0.032; P = 0.032). Other ECG abnormalities such as right ventricular hypertrophy, T-wave changes, and T-axis abnormalities were more prevalent among subjects (10.7%, 5.4%, and 10.7%, respectively) than controls (1.8%, 3.6%, and 14%, respectively), although not significant. **Conclusion:** Electrocardiographic abnormalities are quite prevalent among HIV-infected children, with LVH being predominant. It is recommended that routine ECG evaluations be done on HIV-infected children to enable the early detection and prompt management of these problems.

Keywords: Cardiovascular, electrocardiography, human immunodeficiency virus infection

Introduction

Human immunodeficiency virus (HIV) infection is a disease of major public health significance. It causes significant morbidity and mortality among individuals, particularly children.[1] Globally, about 36.9 million people are estimated to be living with HIV,[2,3] with over half (69.9%) in sub-Saharan Africa.[3] Nigeria has the second highest burden of HIV/AIDS worldwide.[4] In Nigeria, in 2017, 220,000 children aged below 15 years were living with HIV.[4]

With the advent of highly active antiretroviral therapy (HAART), life expectancy of HIV-infected children has increased. This has led to the emergence of late complications of the disease involving the cardiovascular, pulmonary, and neurologic systems.[5,6] The cardiovascular complications of HIV infection affect about 50% of HIV-infected children and has been documented to be a leading cause of morbidity and mortality in HIV-infected children.[6-8] These complications are of major clinical importance as they are mediated by both the HIV and the effects of HAART.[6] Cardiovascular complications have been observed to begin early in the course of the disease

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but may remain asymptomatic until later in life when they present with debilitating and life-threatening symptoms.[9-11] These complications include cardiac arrhythmias, low QRS voltages, conduction defects such as prolonged QTc, varying degrees of atrio-ventricular block, dilated cardiomyopathy, left ventricular systolic dysfunction, left ventricular dilatation, pulmonary hypertension, myocardial infarction, pericardial disease, and heart failure.[12,13] This underscores the need for regular cardiovascular evaluation of HIV-infected children. Unfortunately, in resource-limited countries such as Nigeria, cardiovascular evaluation is yet to be added to the management protocol of pediatric HIV infection.[12]

The electrocardiogram (ECG) remains an invaluable screening tool in the early diagnosis of several cardiac abnormalities. It is a cheap, readily available and does not require so much expertise.[14] Information from the ECG can inform the need for life-saving medical intervention as well as guide the physician in requesting for more detailed and sophisticated investigations such as echocardiography. Globally, studies have shown that ECG abnormalities in HIV-infected children are the independent predictors of incident cardiovascular disease.[14] In Nigeria, a few studies have been done on HIV associated cardiovascular disorders,[15] most of which focused on echocardiographic analysis.[12,13] Unfortunately, echocardiography is not readily available in Nigeria, and where available, it is expensive and requires expertise. In the face of dwindling financial support from nongovernmental organisations for HIV-infected individuals including children, most of these patients who belong to the middle and lower social class[16] can barely afford basic and affordable investigation such as ECG and definitely not the luxury of echocardiographic evaluations. However, little is known about ECG findings among HIV-infected children in Nigeria.[15] The limited studies available have interpreted results using Caucasian ECG normograms as a standard for comparison.[17,18] These Caucasian standards have been shown to differ from that of Africans in several ways.[18]

Using normal ECG standards from Nigerian children, this study aimed to determine the prevalence and types of cardiac abnormalities in HIV-infected children seen in Federal Medical Centre (FMC), Umuahia, using ECG.

Patients, Materials and Methods

This was a hospital-based, comparative, cross-sectional study carried out at Federal Medical Centre, Umuahia, between June and November 2018. The study involved two groups of participants: the HIV-infected group and the HIV-uninfected group. As at the time of the study, a total of 280 HIV-infected children were enrolled in the pediatric HIV clinic which runs once a week with, an average weekly attendance of 20 children.

The sample size was calculated using the standard statistical formula for the comparison of proportions in two equally sized groups.[19]

$$n = \frac{([p - [1 - p1] + [1 - p2]) \times \text{Cp, power}}{(p1 - p2)}$$

$$n = \frac{([p - [1 - p1] + p2[1 - p2]) \times \text{Cp, power}}{(p1 - p2)^2}$$

Where n = number of subjects required in each group:

- p1 = proportion of HIV-infected children with ECG abnormalities
- p2 = proportion of HIV-uninfected children with ECG abnormalities
- Cp, power = constant defined by the values chosen for *P* value and the power of the study.

The power of the study was set at 90% and the $P = 0.05$. This gave a Cp, power = 10.5:[19]

$p1 = 0.18$ based on the study in Jos by Ige *et al*.[17]

$p2 = 0.0072$, based on a study carried out in Jos, Nigeria by Yilgwan *et al*.[20]

Thus,

$$n = \frac{([0.18[1 - 0.18] + 0.0072[1 - 0.0072]) \times 10.5}{(0.18 - 0.0072)^2}$$

$$n = \frac{([0.18 \times 0.82] + [0.0072 \times 0.9928]) \times 10.5}{(0.1728)^2}$$

$$n = \frac{(0.1476 + 0.007148) \times 10.5}{0.02986}$$

$$n = \frac{0.15475 \times 10.5}{0.02986}$$

$n = 54.4 \approx 55$

This meant sample size for each group of participants would be 55.

However, this was the sample size calculated for an infinite population, i.e., population higher than 10,000. Considering the finite population from which the study subjects was drawn (280), it was necessary to adjust the required sample size using the following formula:[21]

$$\text{nf} = \frac{n}{1 + (\text{n} / \text{N})}$$

nf = the desired sample size when population is <10,000.

n = the desired sample size when the population is >10,000.

n = the estimate of the population size (the population of HIV-infected children attending the pediatric HIV clinic at FMC Umuahia being 280).

$$\text{nf} = \frac{n}{1 + (\text{n} / \text{N})}$$

$$nf = \frac{55}{1+(55/280)} = \frac{55}{1+0.1964}$$

nf = 45.97 ≈ 46.

Allowing for attrition rate (r) of 10% (0.1), the minimum sample size (n_s) was adjusted with the formula:[21]

$$ns = \frac{nf}{(1-r)}$$

Substituting nf with the calculated sample size of 46 per participant group,

Thus:

$$ns = \frac{46}{(1-0.1)} = 51.1 \approx 52$$

The minimum sample size for each group was 52. However, the researcher recruited 56 subjects per group.

Sampling of study participants

HIV-infected children were recruited by the systematic random sampling. The total number of HIV-infected children enrolled in the pediatric HIV clinic formed the sampling frame (N). The sampling interval (k) was obtained by dividing the sampling frame by the calculated sample size (ns).[21] Thus:

$$k = \frac{n}{ns}$$

$$k = \frac{280}{52} = 5.3 \approx 6$$

Using the pediatric HIV clinic attendance register on each clinic day, subjects were selected at random after determining a starting point by balloting. This was done by writing the digits one to six on separate but identical pieces of paper and folding them neatly. These pieces of paper were put into a small box and thereafter one of the pieces was blindly chosen. Whatever number selected was traced to the corresponding number on the attendance register. The patient with the corresponding number on the attendance register and every sixth child from that point thereafter who met the inclusion criteria for the HIV-infected group were enrolled. Where a selected subject did not meet the inclusion criteria, the next consecutive patient on the register who met the inclusion criteria was enrolled and the sampling interval of six was observed thereafter. This procedure was repeated every pediatric HIV clinic day until the calculated sample size was attained.

The study population comprised 56 HIV-infected children aged between 18 months and 14 years attending the Pediatric HIV clinic of the FMC, Umuahia, and 56 HIV-uninfected children of the same age range attending the Children Outpatient clinic of the FMC, Umuahia. HIV-infected children served as the subjects in this study, whereas the HIV-uninfected children served as the control group, and were age-and gender-matched with the subjects. Subjects already had their retroviral status confirmed either by the polymerase chain reaction or by western blot as verified from their medical records. Relevant information was obtained through questionnaires, medical records, and physical examination. The subjects who met the inclusion criteria were enrolled into this study using the systematic random sampling, whereas the control group were recruited using the convenient sampling method. Inclusion criteria were age between 18 months and 14 years and confirmation of retroviral status. Subjects and controls with a history of congenital or acquired cardiac disease, renal disease, diarrhea and vomiting, intake of antiarrhythmic drugs or drugs with known cardiovascular effects (for example digoxin, quinidine, and phenytoin) or whose physical examination findings were suggestive of underlying cardiac or renal diseases, were excluded from the study.

Informed consent was obtained from the caregiver and verbal assent from children aged 7 years and above before recruitment. Ethical approval for the study was obtained from the Health Research Ethics Committee of the Federal Medical Centre, Umuahia.

Information on baseline WHO clinical stage and current ART was retrieved from the medical records of subjects. Subjects were also examined and categorised into the appropriate WHO HIV clinical stage.[22]

Sociodemographic data of the subjects and controls were obtained and their socio-economic classes were determined using the Oyedeji's classification.[23] Thereafter, their pulse rate was counted manually for 1 min using the right radial pulse.

Electrocardiography

A portable 12 lead SCHILLER CARDIOVIT-AT 102 Electrocardiograph® (Schiller-Reomed AG, Dietikon, Switzerland) was used to determine the presence or absence of cardiovascular abnormalities in them, based on the American Heart Association recommendation.[24] The ECG recordings were done at a paper speed of 25 mm/s and wave amplitudes were measured at a calibration of 10 mm per millivolt. All recordings were manually measured. Reference values of the ECG analysis were based on the normal ECG standards as reported in the study by Kolawole and Omokhodion.[18] An abnormal ECG was described as any ECG with any parameter outside the normal limits for age for the child. Study participants with abnormal ECG findings were referred to the pediatric cardiology clinic of FMC, Umuahia.

Definition of terms

1. Sinus rhythm was determined by the presence of every QRS complex being preceded by an upright P-wave in lead II with P axis within 90° (upright P-wave in lead I and avF)[25]
2. Heart rate was calculated by counting the number of small boxes between two R-waves and dividing 1500 by it[25]
3. P-wave duration was measured from the onset of P-wave to the end of the wave

4. The P-wave amplitude was measured from the isoelectric line to the upper border of the peak of the wave
5. The PR interval was measured from the onset of the P-wave to the beginning of the QRS complex
6. The QRS complex duration was measured from the onset of the Q-wave (or R wave if no Q wave was visible) to the end of the S-wave
7. The QRS axis was calculated using the hexaxial reference system, using leads I and avF. Normal axis was set at 0°–90°; right axis deviation was set at >90°–180°; left axis deviation was set at − 90° to <0°; indeterminate axis was set at −180° to >-90°[26]
8. The QTc interval was measured by first measuring the QT interval and correcting for participant's heart rate using the Bazett's formula:[25] $QTc = QT/\sqrt{RR}$. Normal QT_c interval was set at 360–460 ms.[25] QT interval was measured from the beginning of the Q-wave to the end of the T-wave. Prolonged QTc was regarded as QTc >460 ms
9. The ST segment was measured in millimetres as elevation or depression from the isoelectric line
10. Lead independent parameters including heart rate, *P* wave amplitude and duration, PR interval, QTc interval, QRS duration and axis, and T waves were assessed using lead II
11. ST segment was assessed on both the chest and limb leads. Any value >1 mm in the limb leads and >2 mm in the chest leads above or below the isoelectric line was considered abnormal
12. Measurements of wave amplitudes were made using PR segment as the isoelectric line (baseline)[25]
13. Left ventricular hypertrophy (LVH) was assessed using the voltage criteria: R wave amplitude >98th percentile for age in lead V6 and an S wave depth >98th percentile for age in lead V1[27,28]
14. Right ventricular hypertrophy (RVH) was defined as R-wave in V1 >98th percentile for age[27,29]
15. Premature ventricular contraction (PVC) was determined as the presence of a widened bizarre QRS complex not preceded by a P-wave appearing earlier than expected with a full compensatory pause following the contraction.[25,30]

Data analysis

Data analysis was performed using the IBM Statistical Package for the Social Sciences (SPSS) for windows software version 20 (IBM Corp., Armonk, N.Y.,USA). Descriptive statistics such as mean and standard deviation were used to analyse the continuous variables. Frequencies and percentages of categorical variables were determined. The categorical variables were compared using the Chi-square test (or Fisher's exact test when a cell's value was <5), while means of continuous variables were compared using Student's *t*-test. Statistical significance was set at $P < 0.05$.

Results

General description of the study population

A total of 112 children comprising 56 HIV-infected subjects and 56 age- and gender-matched controls were enrolled into the study. There were 27 (48.2%) males and 29 (51.8%) females per participant group with a male-to-female ratio of 1:1.07. Ages of participants ranged between two years and 14 years, with a mean age of 8.9 ± 3.5 years and a median age of nine years. Table 1 shows the distribution of study participants according to the age groups.

General description of HIV-infected subjects

WHO clinical stage of subjects at baseline and at recruitment

At baseline, 33.9%, 53.6%, 10.7%, and 1.8% of subjects had asymptomatic, mild, advanced, and severe HIV disease, respectively. A general improvement in disease severity at recruitment was observed as 76.8% of subjects had asymptomatic disease, 21.4% had mild disease, 1.8% had advanced disease, and none had severe disease [Figure 1].

Antiretroviral therapy history of subjects

Fifty (89.3%) children were on HAART, while 6 (10.7%) were HAART-naïve. Subjects on HAART were all on nevirapine, lamivudine, and zidovudine.

Electrocardiogram abnormalities among subjects

ECG abnormalities were observed to be more prevalent among HIV-infected subjects (42.9%, 24/56) than controls (17.9%, 10/56) [$P = 0.004$, odds ratio = 3.45, confidence interval = 1.45–8.19, Table 2].

Table 3 shows the frequency of the different ECG abnormalities documented in the HIV-infected and uninfected participants. LVH, RVH, and abnormal T-axis were more frequent among the HIV-infected children. LVH was however significantly

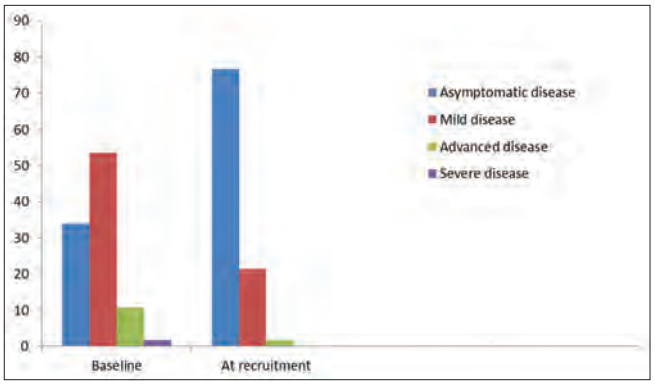

Figure 1: WHO clinical staging of subjects at baseline and at recruitment

Table 1: Demographic characteristics of the study participants (*n*=56)

Age group (years)	Subjects		Controls	
	Male, *n* (%)	Female, *n* (%)	Male, *n* (%)	Female, *n* (%)
<5	0	6 (20.7)	0	6 (100)
5–9	16 (59.3)	10 (34.5)	16 (59.3)	10 (34.5)
10–14	11 (40.7)	13 (44.8)	11 (40.7)	13 (44.8)
Total	27 (100)	29 (100)	27 (100)	29 (100)

Table 2: Comparison of prevalence of electrocardiogram abnormalities among subjects and controls (*n*=56)

ECG findings	HIV-infected (subjects), *n* (%)	HIV-uninfected (controls), *n* (%)	χ^2	*P*	OR (95% CI)
Normal ECG	32 (57.1)	46 (82.1)	8.28	0.004*	3.45 (1.45–8.19)
Abnormal ECG	24 (42.9)	10 (17.9)			

*Significant. OR: Odds ratio, CI: Confidence interval, ECG: Electrocardiogram, HIV: Human Immunodeficiency Virus

Table 3: Comparison of electrocardiogram abnormalities between subjects and controls (*n*=56)

ECG abnormality	HIV-infected (subjects), *n* (%)	HIV-uninfected (controls), *n* (%)	*P*[a]	OR (95% CI)
Rhythm disturbances				
PVC	1 (1.8)	1 (1.8)	1.00	1.00 (0.06–16.39)
Conduction abnormalities				
First degree AV block	1 (1.8)	0	1.00	N/A
Short PR interval	1 (1.8)	0	1.00	N/A
Prolonged QTc	1 (1.8)	0	1.00	N/A
Mean vector (QRS)				
LAD	3 (5.4)	1 (1.8)	0.62	3.11 (0.31–30.88)
RAD	1 (1.8)	1 (1.8)	1.00	1.00 (0.06–16.39)
Chamber enlargement				
LVH	8 (14.3)	1 (1.8)	0.032*	9.17 (1.11–75.96)
RVH	6 (10.7)	1 (1.8)	0.11	6.60 (0.77–56.74)
Abnormal T axis	6 (10.7)	4 (7.14)	0.74	1.56 (0.42–5.86)
T wave change	3 (5.4)	2 (3.6)	1.00	1.53 (0.25–9.52)

*Significant, [a]Fisher's exact test. PVC: Premature ventricular contraction, AV: Atrio-ventricular, QTc: Corrected QT, LAD: Left-axis deviation, RAD: Right-axis deviation, LVH: Left ventricular hypertrophy, RVH: Right ventricular hypertrophy, N/A: Not applicable, OR: Odds ratio, CI: Confidence interval, ECG: Electrocardiogram, HIV: Human Immunodeficiency Virus

more prevalent among subjects than controls ($P = 0.032$). There was no statistical difference in the frequency of occurrence of other ECG abnormalities between HIV infected and uninfected children.

Discussion

The prevalence of ECG abnormalities of 42.9% observed among HIV-infected subjects in this study is comparable to those of Diógenes *et al*.[7] in Brazil and Badal *et al*.[9] in India**,** who documented prevalence rates of 48.8% and 47%, respectively. It was however lower than the finding of Herdy *et al*.[31] in Brazil who documented a prevalence rate of 59.6%. This difference in findings could be due to the differences in the clinical characteristics of the subjects in both studies. In the index study, subjects with a recent history of diarrheal illnesses were excluded from the study. This was to exclude the effect of dyselectrolytemia, especially hypokalemia on the heart as potassium is known to play a key role in repolarisation of the heart. About 48.8% of the subjects in the study by Herdy *et al*.[31] had diarrhea and this could have affected the findings as the most common ECG abnormality detected was ventricular repolarisation changes.

The prevalence of electrocardiographic changes in HIV-infected children was significantly higher than in the controls, and the odds of having ECG abnormalities were three times more in the HIV-uninfected children than controls. This was in consonance with the findings from several studies.[17,32] The higher prevalence among subjects could be attributed to the direct toxic effect of the virus on the heart, as well as the untoward effects of HAART.[33]

Of the several ECG abnormalities noted among the HIV-infected study participants, LVH was the most common (14.3%). Lipshultz *et al*.[10] demonstrated a higher prevalence of LVH of 37%. Lubega *et al*.[8] in Uganda however documented a lower prevalence of LVH of 1.3%. The differences observed might have been due to the choice of criterion used for LVH in the index study. Several ECG criteria exist for LVH.[34] These criteria have been documented to have varying sensitivities.[34] The studies by Lipshultz *et al*.[10] and Lubega *et al*.[8] however, did not state the criteria used for LVH. The criteria for LVH used in this study were R-wave amplitude > 98th percentile for age in lead V6 and an S wave depth > 98th percentile for age in lead V1.[27,28]

LVH was found to be significantly more prevalent among the HIV-infected children (14.3%) than controls (1.8%). A similar observation was documented in the study Attamah *et al*.[33] in Benin, Nigeria, which noted a significant prevalence of LVH among HIV-infected children (8.5%) compared to controls (1.5%) ($\chi^2 = 10.32$; $P = 0.002$). LVH in HIV has been linked to the direct effects of the virus on cardiac myocytes. The virus causes the release of cytokines which leads to myocardial inflammation.[35] LVH has been documented to be an independent predictor of adverse cardiovascular events such as sudden cardiac death and ventricular arrhythmias in HIV-infected children.[35] It has been suggested to be a possible indicator of the beginning of cardiomyopathy later in life.[33,35]

HIV-infected children are thus more at risk for these events compared to HIV-uninfected children. This emphasises the import of regular cardiovascular assessments of HIV-infected children using ECG.

The prevalence of RVH of 10.7% among subjects in this study was similar to the prevalence of 13% documented by Lipshultz *et al*.[10] Predisposition of HIV-infected patients to multiple opportunistic lung infections which are risk factors for pulmonary hypertension and attendant RVH[36] may be the explanation to this finding.

Autonomic dysfunction due to prolonged immunodeficiency and malnutrition has been identified as the possible causes of rhythm disturbances in HIV.[37] The prevalence of PVCs among HIV-infected children in the study was 1.8% and this is comparable to the studies by Herdy *et al*.[31] in the USA and Lubega *et al*.[8] in Uganda who described the prevalence rates of 2.1% and 0.4%, respectively. A higher prevalence of 10% and 30% was however documented in the USA by Luginbuhl *et al*.[11] and Lipshultz *et al*.,[10] respectively. This disparity in findings might be attributed to the cross-sectional design of the index study as only one electrocardiographic evaluation was done per subject. The studies by Luginbuhl *et al*.[11] and Lipshultz *et al*.[10] were prospective studies and subjects had serial ECG analysis as well as 24-h Holter studies performed on them, thus increasing the chances of detecting ECG abnormalities.

Prolonged QTc interval is a risk for life-threatening arrhythmias and sudden death.[30] Prolonged QTc interval was noted in 1.8% of the subjects in this study. This is comparable to the prevalence of 3.5% documented by Attamah *et al*.[33] in Benin. However, it was much lower than the prevalence rates of 18% and 20% documented by Ige *et al*.[17] in Jos, Nigeria and Apsari *et al*.[38] in Indonesia. In this study, prolonged QTc was defined as QTc interval >0.46 s. Apsari *et al*.[38] however defined prolonged QTc as a QTc interval above 0.44 s. This difference in standards may account for the disparity in findings. The difference in the clinical characteristics of the subjects in the extant study and the study by Ige *et al*.[17] might have accounted for this contrasting findings. In the index study, 1.8% of subjects had advanced disease (WHO clinical stage 3), whereas 17% had such severity in the study by Ige *et al*.[17] Studies have shown that worsening autonomic dysfunction occur with advancing HIV disease.[6] Dysautonomia increases the risk for prolonged QTc interval. Thus, the study by Ige *et al*.[17] had more subjects who were at risk for prolonged QTc compared to the index study.

PVCs was observed in 1.8% of participants in the extant study, and this was close to the findings by Herdy *et al*.[31] in Brazil and Lubega *et al*.[8] in Uganda, of 2.1% and 0.4% respectively. However, higher prevalence rates of 30% and 15% were documented by Lipshultz *et al*.[10] and Luginbuhl *et al*.[11] in the USA, respectively. Unlike the studies by Lipshultz *et al*.[10] and Luginbuhl *et al*.,[11] this study did not involve the use of Holter monitoring of subjects; hence, it could account for the dissimilarity in findings. PVCs have been implicated in increasing the risk for heart failure, cardiomyopathy, and sudden death.[39] Thus, early detection and intervention especially in patients with symptomatic PVCs will aid in preventing these debilitating complications.

T-wave changes have been noted to be indicators of possible underlying inflammation of the myocardium.[25] HIV is known to induce a chronic inflammatory response that result in the release of inflammatory biomarkers that lead to myocardial dysfunction.[40] This study observed a 5.4% prevalence rate of T-wave changes among subjects which was close to the prevalence rate of 4.6% documented by Namuyonga *et al*.[41] in Uganda.

First degree block was observed in one (1.8%) HIV-infected participant in this study. First-degree atrio-ventricular AV blocks are a result of prolongation of the PR interval. In Benin, Nigeria, Attamah *et al*.[33] demonstrated a 5% rate of prolonged PR among HIV-infected participants, although the degree of AV block was not specified. It has been documented that prolonged PR interval increases the risk for atrial fibrillation. One subject (1.8%) was found to have a short PR interval in this study. However, there were no findings suggestive of preexcitation syndromes.

Right-axis deviation and left-axis deviation were seen in 1.8% and 5.4% of subjects, respectively. There is a paucity of data on axis deviation abnormalities in children with HIV infection. However, it is well documented that these findings are indicative of abnormalities in the ventricular depolarisation.[42] Such abnormalities include ventricular hypertrophy, bundle branch blocks, and ventricular ectopic rhythms.[25]

There were no significant differences noted in the prevalence rates of RVH, prolonged QTc, T-wave changes, and conduction abnormalities in HIV-infected and HIV-uninfected children. This could be as a result of the sample size as well as disease severity of study participants.

A limitation identified in this study is the sample size. A larger sample size might have impacted on the spectrum and prevalence of different ECG abnormalities detected in the study. However, the small number of HIV-infected children registered in the pediatric HIV clinic as at the time of this study made it a challenge.

Conclusion

The study showed that HIV-infected children are predisposed to a significant degree of electrocardiographic anomalies which portend a high risk for debilitating morbidity and mortality. Hence, it is recommended that routine electrocardiographic assessments be included in the management protocol of these children.

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Conflicts of interest

There are no conflicts of interest.

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