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# Incidence and Evolutionary Trajectory of Left Ventricular Hypertrophy in People Living with HIV in the DRC: A Retrospective Cohort

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## Abstract

**Background:** Left ventricular hypertrophy (LVH) is an early marker of cardiac involvement and a predictor of heart failure and mortality. This study aimed to determine the incidence of LVH, describe its progression trajectory after initiation of antiretroviral therapy (ART), and identify risk factors for LVH among people living with HIV (PLHIV). **Methods:** This retrospective cohort study included 154 adults living with HIV who were followed at the University Clinics of Kinshasa between 2000 and 2003 and had been on ART for at least six months. Clinical, laboratory, and electrocardiographic data were analyzed to assess the incidence, progression, and risk factors of LVH. **Results:** The overall prevalence of LVH was 29.2%. Survival analysis showed that patients on TDF + 3TC + EFV had a higher risk of developing LVH compared to those on TDF + 3TC + LPV/r (adjusted hazard ratio [aHR] = 3.39; p = 0.003). Hypertension (aHR = 3.79; p = 0.001) and proteinuria (aHR = 2.97; p = 0.026) were identified as independent risk factors, while CD4 count was not significantly associated after adjustment. **Conclusion:** These findings confirm the importance of hypertension and renal impairment in left ventricular remodeling and suggest a potential role of ART regimen choice in cardiovascular health.

## Subject Areas

Evolutionary Studies

## Keywords

Left Ventricular Hypertrophy, HIV, Risk Factors, Antiretroviral Therapy,

Democratic Republic of the Congo

## 1. Introduction

Cardiovascular diseases (CVDs) are an increasing cause of morbidity and mortality in sub-Saharan Africa, in a context of epidemiological transition characterized by the coexistence of infectious and non-communicable diseases [1] [2]. Despite this growing burden, few data are available to describe the combined impact of traditional risk factors and endemic factors, including chronic infections such as HIV, on the incidence of cardiovascular complications in this region. Left ventricular hypertrophy (LVH) is a well-established marker of preclinical cardiovascular disease. Detected via electrocardiography and echocardiography, it is strongly associated with an increased risk of heart failure, arrhythmias, and sudden cardiac death [3] [4]. Recent cross-sectional studies in Africa have shown a strong association between LVH, hypertension, and HIV infection, suggesting a complex interplay of these factors in the development of cardiac damage [5] [6]. To date, data describing the incidence and progression trajectory of LVH in sub-Saharan Africa remain scarce, if not entirely absent in some settings. This gap is particularly concerning in a region that houses the majority of people living with HIV (PLHIV), according to UNAIDS [7]. In high-income countries, HIV infection has been associated with an approximately twofold higher risk of developing heart failure compared to the general population [8] [9]. Additionally, cross-sectional studies have reported a higher prevalence of left ventricular dysfunction among PLHIV compared to uninfected individuals, suggesting a direct or indirect effect of HIV on myocardial structure and function [8] [10].

In the Democratic Republic of the Congo, where access to ART has improved significantly in recent years, data on cardiovascular complications among PLHIV remain limited. The objectives of this retrospective cohort study conducted in the DRC were therefore: 1) to determine the incidence of LVH in PLHIV; 2) to describe its progression trajectory after ART initiation; and 3) to explore the impact of traditional cardiovascular risk factors, endemic conditions, and HIV-specific factors on the occurrence and progression of LVH.

## 2. Patients and Methods

### 2.1. Study Design and Population

This was a historical cohort study conducted at the University Clinics of Kinshasa (CUK), a reference and teaching hospital in the DRC, from 2000 to 2025. The study population included adult patients living with HIV, followed at CUK, who had been receiving ART for at least six months. Inclusion criteria were: age ≥18 years, confirmed HIV infection according to national algorithms, and complete clinical, laboratory, and ECG data. Patients with known congenital heart disease, severe rheumatic valvular heart disease, or documented ischemic heart disease

prior to ART initiation, as well as those with incomplete records, were excluded. A total of 231 patient records were initially screened from the health facility registers and databases. Among these, 77 records were excluded due to incomplete or missing key variables (such as demographic information, clinical characteristics, or follow-up data), resulting in a final analytic sample of 154 participants. Regarding missing biological data, particularly viral load and other laboratory values, we adopted the following approach: records with completely missing viral load measurements at baseline or during follow-up were excluded from analyses requiring this variable. For other laboratory parameters with partial missingness, we performed a complete-case analysis, including only observations with available data for the variables of interest. No imputation methods were applied due to the extent and pattern of missing data.

### 2.2. Data Collection

Data were collected from medical records and supplemented by standardized interviews and systematic clinical examination. Clinical variables included sociodemographic characteristics (age, sex), medical and cardiovascular history, including hypertension, diabetes, smoking, and alcohol consumption, as well as clinical parameters such as blood pressure, heart rate, waist circumference, and body mass index. HIV-related data included duration of infection and ART regimen. Laboratory assessment included CD4 lymphocyte count, plasma HIV viral load when available, hemoglobin level, and standard biochemical parameters such as serum creatinine, fasting glucose, and lipid profile. Laboratory abnormalities were defined according to the standards of the University Clinics of Kinshasa and current international guidelines. All patients underwent an ECG performed by an experienced cardiologist, in accordance with the recommendations of the European Society of Cardiology and the American Society of Echocardiography [11]. In this study, LVH was defined based on electrocardiographic (ECG) criteria. Specifically, the diagnosis relied on the Sokolow-Lyon index, calculated as the sum of the S wave amplitude in lead V1 and the R wave amplitude in lead V5 or V6 (whichever was larger). LVH was considered present when this sum was ≥35 mm. ECG was the sole modality used for LVH assessment in this study, as echocardiographic data were not systematically available in the study setting. All ECG recordings were performed using standard 12-lead techniques and interpreted by trained clinicians according to established guidelines.

### 2.3. Statistical Analysis

Data were entered and analyzed using SPSS for Windows, version 25. Continuous variables were expressed as means ± standard deviation or medians with interquartile ranges depending on distribution, and categorical variables were presented as counts and percentages. Statistical comparisons were performed using Student's t-test or Mann-Whitney U test for continuous variables and $\chi^2$ or Fisher's exact test for categorical variables. The temporal progression of LVH was

described using Kaplan-Meier curves, and the Log-rank test was used to compare the different curves. Risk factors for LVH were investigated using a stepwise approach with Cox regression analysis, calculating hazard ratios (HR) and adjusted hazard ratios (aHR) to estimate these associations. In the multivariable analysis, the following candidate covariates were considered based on clinical relevance: HTA, CD4 cell count, proteinuria, and therapy regimen. Variable selection was performed using a stepwise approach. Specifically, variables with a p-value < 0.05 in univariate analysis were entered into the multivariable Cox model, followed by a backward stepwise elimination procedure, retaining variables with a significance level of $p < 0.05$ in the final model. The proportional hazards assumption was assessed using both graphical methods (log-minus-log survival plots) and statistical tests based on Schoenfeld residuals. No significant violations of the proportional hazards assumption were observed.

The study was conducted in accordance with the principles of the Declaration of Helsinki, ensuring confidentiality of data and obtaining informed consent from all participants.

## 3. Results

The study population had a mean age of 50.6 years ± 12 years, with no significant difference between the two ART regimens ($p = 0.884$). Age and sex distribution were comparable across groups, indicating good baseline demographic homogeneity. Clinically, some manifestations differed significantly according to the ART regimen. Patients on TDF + 3TC + LPV/r more frequently reported physical asthenia (17.6% vs. 3.6%, $p = 0.041$), whereas chest pain and exertional dyspnea were significantly more frequent in patients on TDF + 3TC + EFV ($p = 0.031$ and $p = 0.029$, respectively). Angina was observed only in the EFV group. Infectious history showed notable differences: tuberculosis was significantly more frequent among patients treated with EFV (23.4% vs. 5.9%, $p = 0.008$), potentially reflecting clinical considerations or drug interactions influencing regimen choice. The history of hepatitis, alcohol consumption, and smoking did not differ significantly between groups. No significant differences were observed regarding anthropometric parameters (BMI, waist circumference) or hemodynamic parameters (SBP, DBP, pulse pressure), indicating a generally comparable baseline cardiovascular load between regimens (Table 1).

Hematological (hemoglobin) and renal parameters (serum creatinine) were similar between the two groups, suggesting comparable renal tolerance of the therapeutic regimens. In contrast, blood urea levels were significantly higher among patients receiving TDF + 3TC + LPV/r ($p = 0.018$), which may reflect greater metabolic impairment or a more pronounced catabolic state in this group. The lipid profile (total cholesterol, LDL, HDL) showed no significant differences between the two regimens, despite overall elevated LDL levels and low HDL levels, suggesting an increased cardiovascular risk across the entire study population. Glycemic and immunovirological parameters (CD4 count, viral load) were compara-

ble between groups, indicating similar virological and immunological control regardless of the treatment regimen. In contrast, inflammatory markers were significantly higher in patients receiving LPV/r, with increased white blood cell counts ($p = 0.028$) and C-reactive protein levels ($p = 0.014$), reflecting a more pronounced inflammatory state in this group. The erythrocyte sedimentation rate also tended to be higher, although this did not reach statistical significance. Proteinuria was common but similar in both groups (Table 2).

Table 1. Clinical characteristics of patients according to antiretroviral therapy regimen.

| Variable | Over All (n = 154) | TDF + 3TC + EFV (n = 137) | TDF + 3TC + LPV/r (n = 17) | p |
|---|---|---|---|---|
| Age | 50.6 ± 12.0 | 50.9 ± 11.6 | 48.0 ± 15.1 | 0.884 |
| <40 years | 29 (18.8) | 25 (18.2) | 4 (23.5) | |
| 40 - 59 years | 94 (61.0) | 84 (61.3) | 10 (58.8) | |
| ≥60 years | 31 (20.1) | 28 (20.4) | 3 (17.6) | |
| Sex | | | | 0.229 |
| Male | 81 (52.6) | 74 (54.0) | 7 (41.2) | |
| Female | 73 (47.4) | 63 (46.0) | 10 (58.8) | |
| Physical asthenia | 8 (5.2) | 5 (3.6) | 3 (17.6) | **0.041** |
| Chest pain | 20 (13.0) | 19 (13.9) | 1 (5.9) | **0.031** |
| Angina pectoris | 7 (4.5) | 7 (5.1) | 0 (0.0) | - |
| Exertional dyspnea | 50 (32.5) | 46 (33.6) | 4 (23.5) | **0.029** |
| Cardiac palpitations | 22 (14.3) | 20 (14.6) | 2 (11.8) | 0.549 |
| Hypertension | 35 (22.7) | 30 (21.9) | 5 (29.4) | 0.335 |
| Blood transfusion | 22 (14.3) | 51 (15.3) | 1 (5.9) | **0.026** |
| Tabacco consumption | 9 (5.8) | 9 (6.6) | 0 (0.0) | - |
| Alcohol consumption | 23 (14.9) | 23 (16.8) | 0 (0.0) | 0.054 |
| Tuberculosis | 33 (21.4) | 32 (23.4) | 1 (5.9) | 0.008 |
| Hepatitis | 18 (11.7) | 18 (13.1) | 0 (0.0) | 0.106 |
| SBP (mmhg) | 127.2 ± 26.4 | 128.0 ± 25.8 | 120.9 ± 31.4 | 0.298 |
| DBP (mmhg) | 78.8 ± 15.3 | 79.5 ± 15.0 | 72.9 ± 16.8 | 0.095 |
| PP (mmhg) | 48.4 ± 16.7 | 48.5 ± 16.2 | 47.9 ± 21.3 | 0.903 |
| BMI ($Kg/m^2$) | 23.1 ± 4.8 | 23.2 ± 4.9 | 22.2 ± 3.5 | 0.403 |
| WC (cm) | 89.2 ± 9.1 | 89.4 ± 9.1 | 88.0 ± 9.2 | 0.553 |

BMI: Body mass index; WC: Waist circumference; PP: Pulse pressure; DBP: Diastolic blood pressure; SBP: Systolic blood pressure.

**Table 2.** Biological characteristics according to antiretroviral therapy regimen.

| Variable | Over All (n = 154) | TDF + 3TC + EFV (n = 137) | TDF + 3TC + LPV/r (n = 17) | p |
|---|---|---|---|---|
| Hemoglobin (g/dL) | 10.4 ± 2.9 | 10.4 ± 2.8 | 10.5 ± 3.2 | 0.872 |
| Serum creatinine (mg/dL) | 1.05 (0.99 - 1.40) | 1.05 (1.0 - 1.40) | 1.10 (0.90 - 6.59) | 0.451 |
| Blood urea (mg/dL) | 25.0 (18.4 - 41.1) | 23.0 (18.3 - 37.3) | 33.5 (24.9 - 91.0) | **0.018** |
| Total cholesterol (mg/dL) | 177.5 (149.0 - 200.0) | 177.4 (148.0 - 200.0) | 187.0 (147.8 - 200.8) | 0.518 |
| LDL-c (mg/dL) | 141.0 (112.9 - 168.3) | 140.0 (112.8 - 166.5) | 144.0 (111.7 - 172.1) | 0.716 |
| HDL-c (mg/dL) | 28.0 (19.0 - 45.9) | 27.0 (19.0 - 45.3) | 32.0 (20.0 - 51.5) | 0.385 |
| Fasting blood glucose (mg/dl) | 101.0 (90.0 - 152.0) | 101.0 (90.0 - 152.0) | 111.0 (94.0 - 184.0) | 0.263 |
| CD4 (cells/mm$^3$) | 204.5 (120.0 - 374.5) | 220.0 (123.5 - 390.0) | 143.0 (103.0 - 289.0) | 0.144 |
| HIV viral load (copies/mL) | 510.0 (32.8 - 2625.0) | 500.0 (32.0 - 3000.0) | 1000.0 (46.5 - 2060.0) | 0.566 |
| WBC (cells/mm$^3$) | 6.4 (4.7 - 8.8) | 6.1 (4.6 - 8.6) | 8.7 (6.1 - 10.0) | **0.028** |
| ESR (mm/h) | 77.5 (34.0 - 110.0) | 75.2 (33.0 - 110.0) | 100.0 (72.1 - 114.8) | 0.089 |
| CRP (mg/dL) | 44.5 (18.0 - 91.7) | 40.0 (15.0 - 88.3) | 77.8 (45.0 - 94.4) | **0.014** |
| Proteinuria | 118 (76.6) | 105 (76.6) | 13 (76.5) | 0.937 |

LDL-C: Low-density lipoprotein cholesterol; HDL-C: High-density lipoprotein cholesterol; WBC: White blood cell count; ESR: Erythrocyte sedimentation rate; CRP: C-reactive protein.

### Frequency of Left Ventricular Hypertrophy

The figure shows the distribution of participants according to the presence of left ventricular hypertrophy (LVH). Out of a total of 154 subjects, 45 (29.2%) had LVH, while the majority, 109 (70.8%), did not (Figure 1).

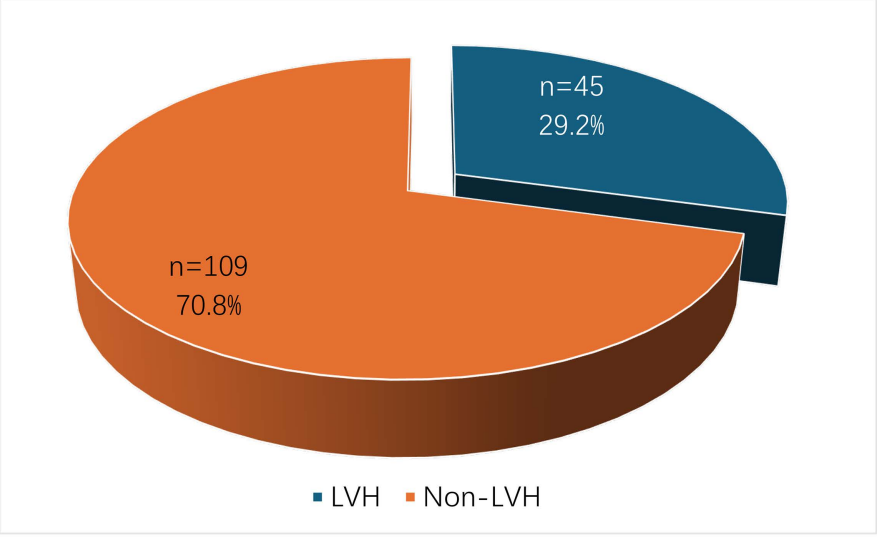


**Figure 1.** Frequency of left ventricular hypertrophy in the study population.

### Evolution of LVH During the One-Year Follow-Up

Survival analysis according to the antiretroviral therapy (ART) regimen showed a notable difference between the two groups. Among the 154 participants, the majority were on TDF + 3TC + EFV (137 subjects), with an LVH proportion of 31.4%, compared to only 11.8% in those on TDF + 3TC + LPV/r. The mean survival time was longer in patients receiving TDF + 3TC + LPV/r (22.52 months) compared to those on TDF + 3TC + EFV (20.04 months) ($p < 0.001$) (Figure 2).

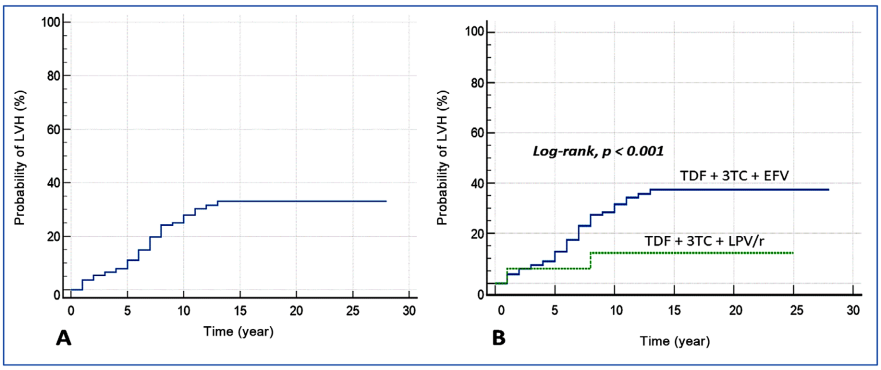


Figure 2. Cumulative temporal evolution of LVH: (A) In the overall population; (B) According to ART regimen.

Survival analysis conducted among 154 participants, of whom 29.2% developed LVH, showed that hypertension (HTN) is associated with LVH. Indeed, the proportion of events was higher in hypertensive subjects (37.1%) than in non-hypertensive subjects (26.9%), with a shorter mean survival time (16.52 vs. 21.25 months). Survival probabilities decreased more rapidly in patients with HTN, as illustrated by the Kaplan-Meier curves, with a statistically significant difference (log-rank $p = 0.001$) (Figure 3(A)).

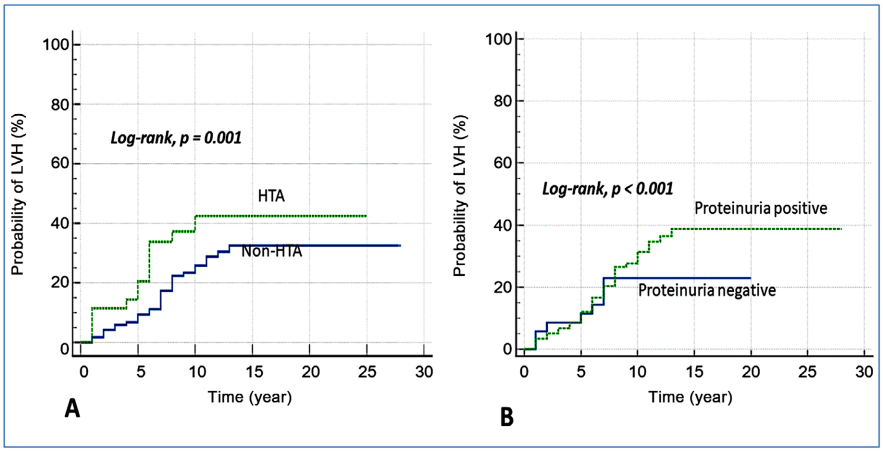


Figure 3. Cumulative temporal evolution of LVH: (A) According to hypertension; (B) According to proteinuria.

Regarding proteinuria, patients with positive proteinuria also showed a higher

event frequency (31.1% vs. 22.9%). However, survival differences between groups were less pronounced, although there was a trend toward more rapid decline in survival among proteinuria-positive subjects, as suggested by the curves (log-rank $p < 0.001$) (Figure 3(B)).

**Risk Factors for LVH Over Time**

In univariate analysis, hypertension (HTN) was strongly associated with the occurrence of LVH, with nearly a fourfold increased risk (HR = 3.72; 95% CI: 1.90 - 6.27; p = 0.001). This association persisted in multivariate analysis (aHR = 3.79; 95% CI: 1.92 - 6.46; p = 0.001). Positive proteinuria was associated with an increased risk of LVH in univariate analysis (HR = 3.15; 95% CI: 1.74 - 6.81; p = 0.006), and this association remained significant after adjustment (aHR = 2.97; 95% CI: 1.69 - 4.74; p = 0.026). Regarding the initial antiretroviral regimen, patients on TDF + 3TC + EFV had a significantly higher risk of developing LVH compared to those on TDF + 3TC + LPV/r, both in univariate analysis (HR = 3.04; 95% CI: 1.74 - 12.54; p = 0.001) and multivariate analysis (aHR = 3.39; 95% CI: 1.82 - 6.08; p = 0.003). In contrast, although CD4 count was associated with LVH in univariate analysis (HR = 1.99; 95% CI: 1.09 - 2.98; p = 0.046), this association disappeared after adjustment (aHR = 1.19; 95% CI: 0.58 - 1.39; p = 0.297) (Table 3).

Table 3. Risk factors for LVH over time.

| Facteurs | Univariate Analysis | | Multivariate Analysis | |
|---|---|---|---|---|
| | p | HR (95% CI) | p | aHR (95% CI) |
| HTA | | | | |
| No | | 1 | | 1 |
| Yes | 0.001 | 3.72 (1.90 - 6.27) | 0.001 | 3.79 (1.92 - 6.46) |
| Proteinuria | | | | |
| Negative | | 1 | | 1 |
| Positive | 0.006 | 3.15 (1.74 - 6.81) | 0.026 | 2.97 (1.69 - 4.74) |
| Therapy regimen | | | | |
| TDF + 3TC + LPV/r | | 1 | | 1 |
| TDF + 3TC + EFV | 0.001 | 3.04 (1.74 - 12.54) | 0.003 | 3.39 (1.82 - 6.08) |
| CD4 (cells/mm$^3$)* | 0.046 | 1.99 (1.09 - 2.98) | 0.297 | 1.19 (0.58 - 1.39) |

*The variable was included in the model as a continuous variable.

## 4. Discussion

This study highlights a substantial frequency of left ventricular hypertrophy (LVH) among people living with HIV (PLHIV), estimated at 29.2%. This proportion is comparable to that reported in other African and international studies, which underscores a high prevalence of cardiovascular complications in this pop-

ulation, related both to the infection itself, chronic inflammation, and the effects of antiretroviral therapy (ART) [3]. Demographically, the homogeneity of the groups in terms of age and sex strengthens the internal validity of comparisons by limiting potential confounding. The relatively high mean age (50.6 years) corresponds to the epidemiological transition observed in PLHIV, characterized by an increase in non-communicable diseases, particularly cardiovascular conditions [1]. The clinical differences observed between ART regimens are particularly noteworthy. The higher frequency of exertional dyspnea, chest pain, and angina in the TDF + 3TC + EFV group suggests more pronounced cardiovascular involvement in this group. Conversely, the higher prevalence of fatigue among patients on LPV/r may reflect metabolic or systemic adverse effects associated with protease inhibitors. Furthermore, the higher prevalence of tuberculosis in the EFV group is consistent with therapeutic guidelines, as efavirenz is often preferred due to better compatibility with antitubercular treatment [12]. The absence of significant differences in anthropometric and hemodynamic parameters suggests that baseline cardiovascular risk was generally comparable between groups. However, the overall unfavorable lipid profile observed in the study population (high LDL, low HDL) confirms the presence of increased cardiovascular risk among PLHIV, as widely documented in the literature [13].

Biologically, elevated inflammatory markers (CRP, white blood cells) in patients on LPV/r suggest a more pronounced inflammatory state, possibly related to the metabolic effects of protease inhibitors. Chronic inflammation is a key mechanism in the development of cardiovascular damage and LVH in PLHIV [3]. Survival analysis revealed a significant difference between ART regimens, with a higher risk of LVH occurrence in patients on TDF + 3TC + EFV. This association was confirmed in multivariate analysis, suggesting an independent effect of the treatment. These findings are consistent with some studies showing an association between certain ART regimens and cardiovascular risk, although data remain sometimes contradictory depending on context and population [14]. Hypertension (HTN) also emerged as the main independent risk factor for LVH, nearly quadrupling the risk. This finding is expected and aligns with pathophysiological knowledge, as HTN is a major determinant of left ventricular remodeling in both the general population and PLHIV [15]. Similarly, proteinuria, a marker of renal impairment and endothelial dysfunction, was independently associated with LVH, highlighting the interaction between renal and cardiovascular damage [15]. In contrast, although CD4 count was associated with LVH in univariate analysis, it was no longer significant after adjustment, suggesting that immunosuppression plays an indirect role, likely mediated by other factors such as inflammation or comorbidities.

This study has some limitations, notably the small size of the LPV/r group, which may limit statistical power and the generalizability of results. Additionally, the observational nature of the study does not allow for causal inference.

## 5. Conclusion

This study demonstrates a high prevalence of LVH among PLHIV and identifies

hypertension, proteinuria, and ART regimen as independent risk factors. These findings underscore the importance of integrated care, including screening and management of cardiovascular risk factors in this population.

## Conflicts of Interest

The authors declare no conflicts of interest.

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