ARAŞTIRMA MAKALESİ / *RESEARCH ARTICLE*

Cardiovascular Effects of Antiretroviral Therapy in Human Immunodeficiency Virus Patients: A Strain Echocardiography Study

İmmün Yetmezlik Virüsü Hastalarında Antiretroviral Tedavinin Kardiyovasküler Etkileri: Bir Strain Ekokardiyografi Çalışması

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ABSTRACT

Aim: *The availability of antiretroviral treatments (ART) has transformed Human Immunodeficiency Virus (HIV) management, turning it from a fatal infection into a manageable chronic condition by effectively reducing viral replication and enhancing patient survival rates. Cardiovascular disease (CVD) is increasingly recognized as a leading cause of morbidity and death among people living with HIV, highlighting the need to monitor potential adverse effects of ART on the cardiovascular system. This study assessed the cardiovascular effects of ART in a cohort of HIV-infected patients by analyzing laboratory, echocardiographic strain, and demographic data before and after treatment.*

Material and Methods: *A total of 56 patients who attended the infectious diseases clinic and were scheduled to receive ART treatment were included in the study. A comprehensive cardiovascular assessment, comprising anamnesis, electrocardiogram, and transthoracic strain echocardiography, was conducted before and three months after the initiation of ART therapy.*

Results: *Examination of lipid parameter changes across treatment regimens revealed a statistically significant elevation in total cholesterol, low-density lipoprotein (LDL) cholesterol, and high-density lipoprotein (HDL) cholesterol in the group treated with tenofovir alafenamide following treatment initiation. Regarding echocardiographic parameters evaluated post-treatment, there was no statistically significant change observed in left ventricular systolic function (LVEF) and diastolic function (septal E/e'ratio) (60.81±5.5 vs. 61.16±4.3, p=0.313, and 9.23±2.2 vs. 9.43±2.6, p=0.214, respectively). Notably, a significant improvement was observed in left ventricular global longitudinal strain (LV-GLS), with values changing from -18.74±2.9 to -19.75±2.4 (p<0.001).*

Conclusion: *Our study demonstrates that ART can exert a beneficial influence on cardiovascular health by enhancing myocardial strain and diminishing inflammatory markers in patients infected with HIV.*

Key words: *antiretroviral treatments; human immunodeficiency virus; strain echocardiography*

ÖZET

Amaç: *Antiretroviral tedavilerin (ART) kullanılabilirliği, İnsan İmmün Yetmezlik Virüsü (HIV) yönetimini dönüştürerek, virüs replikasyonunu etkili bir şekilde azaltıp hasta sağkalım oranlarını artırarak, ölümcül bir enfeksiyondan yönetilebilir kronik bir duruma dönüştürmüştür. Kardiyovasküler hastalık (KVH), HIV ile yaşayan kişilerde morbidite ve ölümün önde gelen nedenlerinden biri olarak giderek daha fazla kabul görmektedir ve bu da ART'nin kardiyovasküler sistem üzerindeki potansiyel olumsuz etkilerinin izlenmesi ihtiyacını vurgulamaktadır. Bu çalışma, HIV enfeksiyonlu bir hasta kohortunda ART'nin kardiyovasküler etkilerini, tedavi öncesi ve sonrası laboratuvar, ekokardiyografik suş ve demografik verileri analiz ederek değerlendirmiştir.*

Materyal ve Metot: *Enfeksiyon hastalıkları kliniğine başvuran ve ART tedavisi alması planlanan toplam elli altı hasta çalışmaya dâhil edilmiştir. Antiretroviral tedavisine başlamadan önce ve üç ay sonra, anamnez, elektrokardiyogram ve strain transtorasik ekokardiyografiyi içeren kapsamlı bir kardiyovasküler değerlendirme yapılmıştır.*

Bulgular: *Çeşitli tedavi rejimlerine göre lipit parametrelerindeki değişikliklerin incelenmesi, tenofovir alafenamid tedavisine tabi tutulan grupta tedavi uygulamasından sonra toplam kolesterolde, düşük yoğunluklu lipoprotein (LDL) kolesterolde ve yüksek yoğunluklu lipoprotein (HDL) kolesterolde istatistiksel olarak anlamlı bir artış olduğunu ortaya koymuştur. Tedavi sonrası değerlendirilen ekokardiyografik parametrelere ilişkin olarak, sol ventrikül sistolik fonksiyonunda (LVEF) ve diyastolik fonksiyonunda (septal E/e oranı) istatistiksel olarak anlamlı bir değişiklik gözlenmemiştir (sırasıyla 60,81±5,5'e karşı 61,16±4,3, p=0,313 ve 9,23±2,2'ye karşı 9,43±2,6, p=0,214). Özellikle, sol ventrikül global uzunlamasına strain'de (LV-GLS) önemli bir iyileşme gözlemlendi; değerler -18,74±2,9'dan -19,75±2,4'e değişti (p<0,001).*

Sonuç: *Çalışmamız, ART'nin HIV ile enfekte hastalarda miyokardiyal gerilimi artırarak ve enflamatuvar belirteçleri azaltarak kardiyovasküler sağlık üzerinde faydalı bir etki gösterebileceğini ortaya koymaktadır.*

Anahtar kelimeler: *antiretroviral tedaviler; insan immün yetmezlik virüsü; strain ekokardiyografi*

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Introduction

The Human Immunodeficiency Virus (HIV) remains a major global health issue, affecting millions of people worldwide. The 2022 United Nations Programme on HIV/AIDS (UNAIDS) report states that HIV has negatively impacted human health and caused socioeconomic problems across the globe (1). The availability of antiretroviral treatments (ART) has changed the way HIV is managed, turning it from a deadly disease into a manageable chronic condition by effectively reducing viral replication and increasing patient survival rates (2).

Although antiretroviral therapy (ART) offers important benefits, it is also associated with some non-AIDS-related complications. Cardiovascular disease (CVD) is increasingly recognized as a leading cause of morbidity and death in people living with HIV, highlighting the need to monitor potential adverse effects of ART on the cardiovascular system (3,4). Research indicates that ART may contribute to cardiovascular problems through metabolic changes, endothelial dysfunction, and ongoing immune activation (5).

In particular, ART regimens that include protease inhibitors or integrase strand transfer inhibitors have been linked to dyslipidemia, insulin resistance, and hypertension (6). Each of these conditions is a well-known risk factor for CVD, and their combined effect may further increase cardiovascular risks. However, untreated HIV infection also carries its own risks due to the virus' s direct influence on inflammatory pathways (7). This chronic inflammation can contribute to cardiovascular issues such as myocardial dysfunction and atherosclerosis. Therefore, understanding how ART and untreated HIV influence cardiovascular health is essential for improving patient outcomes (8).

This study assessed the cardiovascular effects of ART in a cohort of HIV-infected patients by analyzing laboratory results, echocardiographic strain data, and demographic information before and after therapy.

Material and Method

After receiving approval from the ethics committee, sixty-five HIV patients who visited the infectious diseases clinic and planned ART treatment were enrolled in the study. Patients aged 18 years or older who intended to start ART, voluntarily agreed to participate, and had optimal echocardiographic and laboratory data were included. Patients with coronary artery disease, LVEF <50%, those refusing ART, or lacking optimal echocardiographic or laboratory data were excluded. After applying exclusion criteria, fifty-six patients were included in the study. Routine cardiovascular assessments and baseline laboratory tests were conducted before treatment. The cardiovascular evaluation included medical history, electrocardiogram, and transthoracic echocardiography. Clinical information on comorbidities, medical history, and current cardiovascular medications was gathered through a thorough review of each patient's medical records and a self-reported questionnaire. Patients were re-evaluated 3 months after initiating ART treatment (Fig. 1).

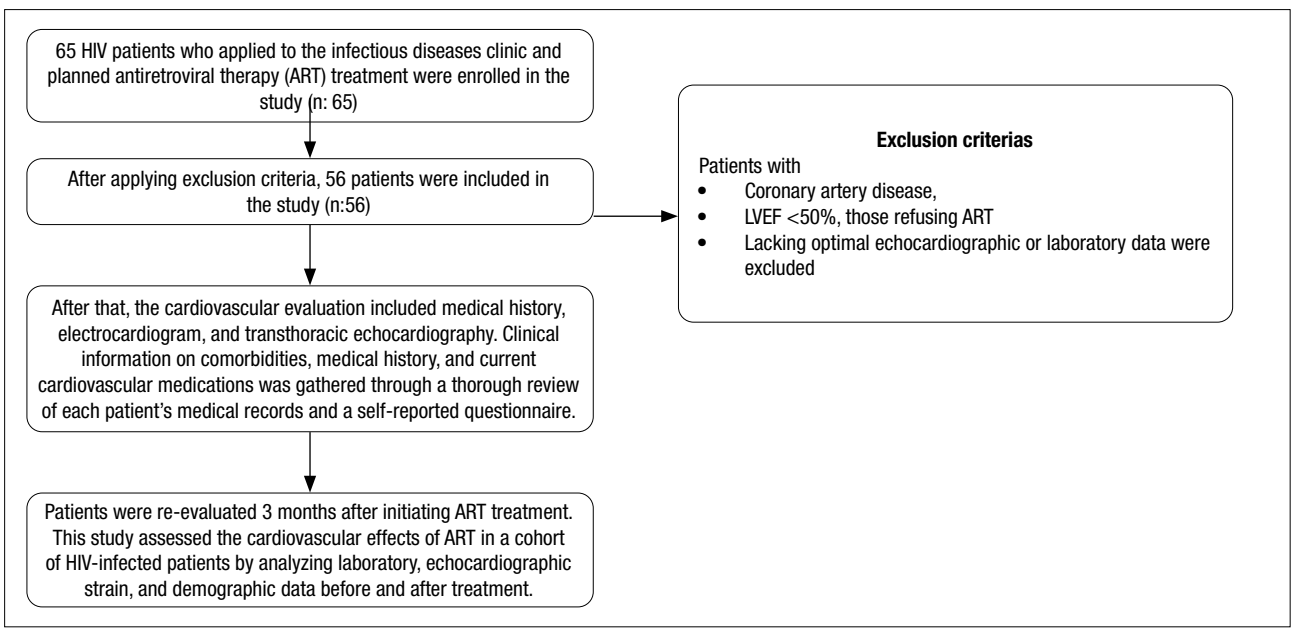

Figure 1. *Flowchart.*

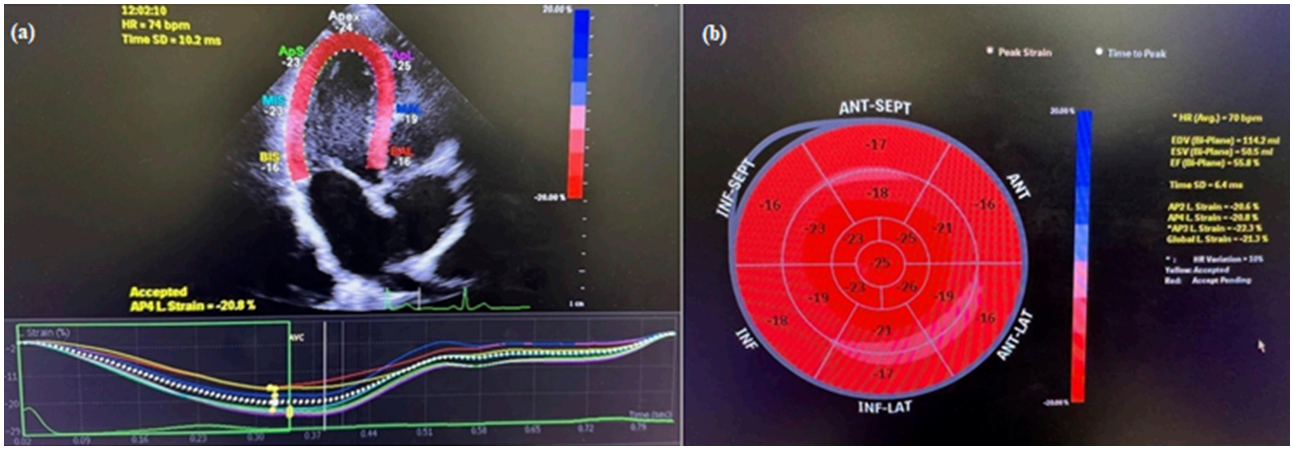

Figure 2. *(a) Systolic longitudinal strain was automatically obtained from the three standard apical views, (b) The average systolic longitudinal strain value from the three apical views was regarded as the global longitudinal strain.*

Echocardiographic Evaluation

Echocardiographic evaluations were conducted using the Philips EPIQ 7 ultrasound system (Philips Medical Systems, USA) equipped with an X5–1 transducer and analyzed offline via Q-Lab software (version 10, Philips Medical Systems, USA). Two independent, experienced cardiologists—blinded to all clinical and outcome-related information—carried out batch measurements of all parameters. The left ventricular ejection fraction (LVEF) was determined by the biplane Simpson's method using apical two– and four-chamber views, with LVEF ≥50% considered preserved. Left ventricular systolic and diastolic diameters were obtained from the parasternal long axis. Mitral inflow velocities—early (E) and late (A)—were assessed using pulsed-wave Doppler. Pulmonary artery systolic pressure (PASP) was estimated based on tricuspid regurgitation velocity (TRV) and right atrial pressure. For 2D speckle-tracking strain analysis, apical two-, three-, and four-chamber views were recorded at frame rates exceeding 50 fps. Strain assessments were performed offline using the Automated Cardiac Motion Quantification feature within Q-Lab 10. The region of interest (ROI) was defined by placing a minimum of 15 manual reference points along the endocardial border in each apical view. Longitudinal systolic strain was computed automatically for each apical view, and the global longitudinal strain (GLS) was calculated as the average of these values (Fig. 2a and 2b).

Statistical analysis

Statistical evaluations were conducted using IBM Statistical Package for Social Sciences (SPSS) program version 15.0 software for Windows (IBM Inc., Chicago, IL, USA). The Kolmogorov-Smirnov test was applied to determine whether continuous data followed a normal distribution. Continuous variables are summarized either as the mean ± standard deviation (SD) or as the median with the minimum and maximum values, depending on the distribution. Categorical data are expressed as frequencies and percentages. For comparisons of continuous variables before and after ART, the paired-samples t-test was used. Differences in categorical variables were assessed using the Pearson chi-square or Fisher's exact test, as appropriate. A p-value below 0.05 was interpreted as indicating statistical significance.

Results

Of the 56 patients included in the study, 49 (87.5%) were male. The mean age of these patients was 40.12±11.9 years. Hypertension was present in 3 (5.4%) patients, and diabetes mellitus was present in 4 (7.1%) patients. All patients were in sinus rhythm on baseline ECG. Twenty-four (42.9%) of the patients were receiving emtricitabine/tenofovir disoproxil + dolutegravir, 13 (23.2%) lamivudine + dolutegravir, and 19 (33.9%) bictagravir/emtricitabine/tenofovir alafenamide regimens (Table 1).

In the laboratory, parameters of the patients were measured before and 3 months after starting the treatment; significant increases were observed in total cholesterol and high-density lipoprotein (HDL) cholesterol levels (164.7±44.3 vs. 177.1±54.1, p=0.01, and 37.6±14.2 vs. 39.8±14.3, p=0.011). Additionally, a significant increase in the CD4 lymphocyte count was noted with treatment (22.19±14.3 vs. 30.15±15.3, p=0.042) (Table 2).

Table 1. *Baseline demographic and laboratory data of patients*

Variables		Patients (n=56)
Age, years (mean ± SD)		40.12±11.9
Male gender, n (%)		49 (87.5)
Hypertension, n (%)		3 (5.4)
Diabetes mellitus, n (%)		4 (7.1)
Hyperlipidemia, n (%)		2 (3.6)
CKD, n (%)		3 (5.4)
CVD, n (%)		1 (1.8)
COPD, n (%)		1 (1.8)
Smoking, n (%)		38 (67.9)
Alcohol, n (%)		40 (71.4)
ECG rhythm, (sinusrhythm), n (%)		56 (100)
ECG heart rate		77.82±12.9
Mild to moderate MR, n (%)		19 (33.9)
Mild to moderate AR, n (%)		3 (5.4)
Mild to moderate TR, n (%)		14 (25)
Treatment regimens	Regime 1	24 (42.9)
	Regime 2	13 (23.2)
	Regime 3	19 (33.9)
Laboratory Data		
FBG, mg/dL		93.41±15.9
Urea, mg/dL		30.67±14.3
Creatinine, mg/dL		0.89±0.28
Uricacid, mg/dL		5.43±1.3
Albumin, g/dL		41.3±5.8
WBC, × 10^9/L		6.59±2.8
Neutrophil count, × 10^9/L		3.94±2.7
Lymphocyte count, × 10^9/L		1.93±0.7
Monocyte count, × 10^9/L		0.53±0.2
Hemoglobin, g/dL		13.75±2.1
Platelet count, × 10^9/L		225.42±63.3
HbA1 c, %		5.41±0.6
CRP, mg/dL		8.52±32.5

SD: standard deviation; n: number of patients; AR: aortic regurgitation; CKD: chronic kidney disease; COPD: chronic obstructive pulmonary disease; CRP: C reactive protein; CVD: cerebrovascular disease; ECG: electrocardiography; FBG: fasting blood glucose; MR: mitral regurgitation; TR: tricuspid regurgitation; WBC: white blood count; Regime 1: emtrisitabin/tenofovirdisoproksil + dolutegravir; Regime 2: lamuvidin+dolutegravir; Regime 3: biktagravir/emtrisitabine/tenofaviralafenamid.

Table 2. *Pre-treatment and post-treatment laboratory data of patients*

Variables (mean ± SD)	Pre-treatment (n=56)	Post-treatment (n=56)	P value
Total cholesterol, mg/dL	164.7±44.3	177.1±54.1	**0.010**
LDL, mg/dL	100.3±37.2	106.9±46.1	0.108
HDL, mg/dL	37.6±14.2	39.8±14.3	**0.011**
Triglyceride, mg/dL	133.7±71.9	153.6±102.9	0.05
CD4 lymphocyte count, mm^3	22.19±14.3	30.15±15.3	0.042
CD8 lymphocyte count, mm^3	48.37±16.7	46.69±12.1	0.634
CRP, mg/dL	8.52±32.5	7.09±13.5	0.611

SD: standard deviation; n: number of patients; CRP: C reactive protein; HDL: high density cholesterol; LDL: low density cholesterol.

When examining changes in lipid parameters based on treatment regimens, a significant increase was noted in total cholesterol, low-density lipoprotein (LDL) cholesterol, and HDL cholesterol in the group treated with tenofovir alafenamide after treatment (164.3±39.1 vs 187.1±34.1, p: 0.010; 100.5±34.7 vs 116.1±34.2, p: 0.039; and 36.1±12.8 vs 40.9±12.9, p: 0.024). In contrast, no significant changes were observed in these lipid parameters in the group that did not receive this treatment (164.8±47.4 vs 172.2±61.6, p: 0.207; 100.2±39.1 vs 102.4±50.9, p: 0.661; 38.4±15.1 vs 39.2±15.2, p: 0.219), as shown in Figure 3a and 3b.

When examining the baseline echocardiographic parameters of the patients, the mean left ventricular ejection fraction (LVEF) was 60.81±5.5, and the mean left ventricular global longitudinal strain (LV-GLS) was -18.74±2.9. Mild to moderate mitral regurgitation was observed in 19 patients (33.9%), mild to moderate aortic regurgitation in 3 patients (5.4%), and mild to moderate tricuspid regurgitation in 14 patients (25%) (Table 1).

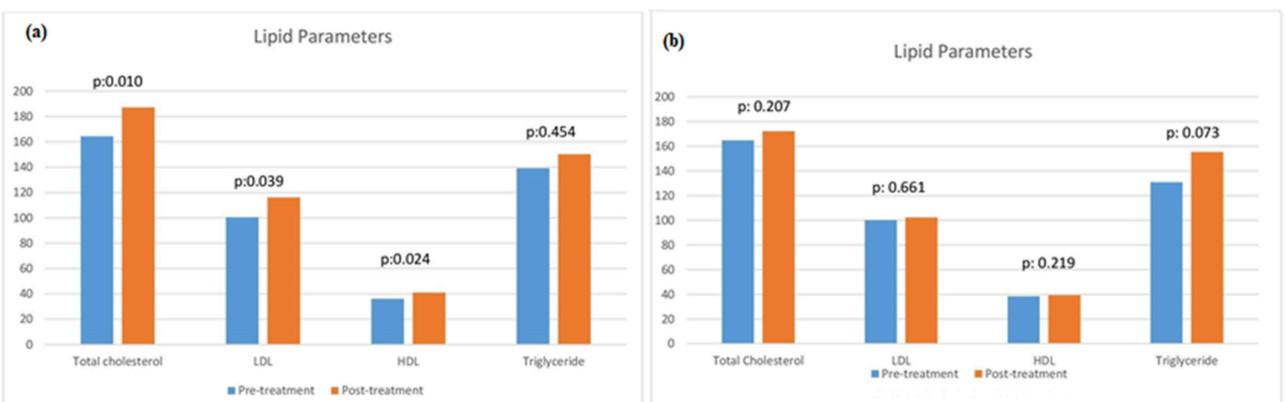

Figure 3. *(a) Post-treatment lipid profile changes in patients receiving tenofoviralafenamide regimen treatment (b) Post-treatment lipid profile changes in patients treated without tenofoviralafenamide regimen.*

Table 3. *Pre-treatmentand post-treatment echocardiography data of the patients*

Variables (mean ± SD)	Pre-treatment (n=56)	Post-treatment (n=56)	P value
Interventricular septum, mm	9.71±1.4	9.73±1.3	0.709
Posterior wall, mm	9.37±1.3	9.37±1.2	1
End-diastolicdiameter, mm	47.17±3.9	47±3.5	0.142
End-systolicdiameter, mm	28.01±4.1	27.87±3.7	0.429
Left atrium diameter, mm	32.96±3.7	33.5±3.3	0.067
Ascending aorta, mm	31.45±4.3	31.56±4.4	0.159
sPAP, mmHg	17±6.1	17.48±4.2	0.277
TAPSE, mm	21.41±1.7	21.75±1.8	0.174
E wavevelocity, cm/s	86.08±16.3	87.69±12.9	0.171
Septale'wavevelocity, cm/s	9.71±2.6	9.78±2.4	0.604
E/Aratio	1.21±0.3	1.22±0.21	0.552
SeptalE/e'ratio	9.23±2.2	9.43±2.6	0.214
Enddiastolicvolume, mL	91.22±15.5	90.14±12.3	0.282
Endsystolicvolume, mL	36.17±9.8	36.22±6.5	0.947
LVEF, %	60.81±5.5	61.16±4.3	0.313
LV-GLS, %	-18.74±2.9	-19.75±2.4	**<0.001**

SD: standard deviation; n: number of patients; LV-GLS: left ventricular global longitudinal strain; LVEF: left ventricular ejection fraction; sPAP: systolic pulmonary artery pressure.

When analyzing the changes in echocardiographic parameters after treatment, there was no significant difference in the LVEF or diastolic functions (septal E/e'ratio) (60.81±5.5 vs 61.16±4.3, p: 0.313 and 9.23±2.2 vs 9.43±2.6, p: 0.214), while a significant increase was observed in LV-GLS with treatment (-18.74±2.9 vs -19.75±2.4, p<0.001) (Table 3).

Discussion

This study highlights the cardiovascular effects of antiretroviral therapy (ART) on left ventricular function in HIV-infected patients, as assessed by strain echocardiography. Our findings suggest that ART may have a positive impact on myocardial strain, supporting the idea that ART alleviates cardiovascular stress caused by untreated HIV infection.

HIV infection itself is linked to an increased risk of cardiovascular disease (CVD), independent of traditional risk factors. Chronic immune activation and systemic inflammation lead to endothelial dysfunction and atherosclerosis, increasing the likelihood of cardiovascular events (9). Antiretroviral therapy has been shown to reduce inflammation, but some regimens, especially protease inhibitors and integrase strand transfer inhibitors, are associated with metabolic issues such as dyslipidemia and insulin resistance (10). Our findings are consistent with previous research showing elevated lipid levels in patients taking tenofovir alafenamide, a known effect of this drug class (6).

A key finding of our study was the notable improvement in left ventricular global longitudinal strain (LV-GLS) following ART initiation. This indicates a possible protective effect of ART on heart function, potentially by lowering systemic inflammation (11). Prior research has shown similar results, suggesting that ART may enhance myocardial strain and decrease subclinical myocardial dysfunction (12). However, the exact mechanisms behind these effects are still not fully understood.

Despite improvements in myocardial strain, we observed no significant changes in LVEF or diastolic function. This suggests that while ART may improve myocardial deformation parameters, it does not necessarily result in substantial enhancements in traditional echocardiographic measures of systolic or diastolic function. Similar findings have been reported in studies using multimodality imaging techniques (13).

Our study also corroborates the growing body of evidence suggesting that HIV-associated CVD risk is multifactorial, involving direct viral effects, chronic inflammation, and ART. Our study also confirms the expanding evidence that HIV-related CVD risk is multifactorial, involving direct viral effects, chronic inflammation, and ART-induced metabolic changes (14). Notably, studies have shown increased arterial inflammation and coronary atherosclerotic plaque burden in HIV-infected patients, even among those with well-controlled viral loads (15). These findings

emphasize the need for comprehensive cardiovascular risk assessment and long-term monitoring in this population. Inflammation and coronary atherosclerotic plaque burden in HIV-infected patients, even in those with well-controlled viral loads (15). These findings highlight the importance of comprehensive cardiovascular risk assessment and long-term monitoring in this population.

Given the metabolic effects associated with certain ART regimens, personalized treatment strategies are crucial to reduce cardiovascular risk while maintaining optimal viral suppression. Approaches such as lipid monitoring, lifestyle changes, and, in some cases, statin therapy, can be helpful (16).

Limitations

Our study has some limitations. The sample size was relatively small, and the follow-up period was limited to three months. Longer-term studies with larger cohorts are necessary to assess the sustained cardiovascular effects of ART. Additionally, while strain echocardiography offers valuable insights into myocardial function, further studies using cardiac magnetic resonance imaging could improve our understanding of myocardial tissue changes (17).

Conclusion

Our study shows that antiretroviral therapy (ART) can positively influence cardiovascular health by improving myocardial strain and decreasing inflammatory markers in HIV-infected patients. These findings support previous research suggesting that ART may reduce cardiovascular strain linked to untreated HIV infection. However, due to potential metabolic side effects from certain ART regimens, ongoing monitoring of cardiovascular health is crucial for optimizing outcomes in HIV management.

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