



Prevalence of Structural Heart Disease Detected By Echocardiography among Adults Living with Human Immunodeficiency Virus on Antiretroviral Therapy in Eastern Nepal

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ABSTRACT:

Background: Improved survival on antiretroviral therapy has shifted morbidity in human immunodeficiency virus infection towards non-communicable cardiac disease, yet echocardiographic data from South Asia remain sparse.

Aims/Objectives: To determine the prevalence of structural heart disease, comprising dilated cardiomyopathy and valvular heart disease, and its association with age, sex and CD4 count.

Methodology: A hospital-based cross-sectional study enrolled 126 consecutive adults on antiretroviral therapy at a tertiary centre in eastern Nepal between August 2018 and July 2019. Those with hypertension, alcohol dependence, cardiotoxic drug exposure or pre-existing cardiac or systemic disease were excluded. All underwent transthoracic echocardiography and CD4 estimation.

Results: Structural heart disease was present in 19 (15.1%) participants: dilated cardiomyopathy in 14 (11.1%) and valvular heart disease in 7 (5.6%). Prevalence did not differ across age groups ($\chi^2 = 0.94$, $p = 0.627$) or by sex (11/64 men, 17.2%, versus 8/62 women, 12.9%; odds ratio 1.40, 95% CI 0.52–3.76, $p = 0.502$). Mean CD4 count was similar in affected and unaffected participants (627 ± 309 versus 551 ± 248 cells/ μ L; $t = 1.18$, $p = 0.239$).

Conclusions: One in seven treated adults had unsuspected structural heart disease, independent of age, sex and CD4 count, supporting routine echocardiographic screening.

Key-words: Cardiomyopathy; Heart valve diseases; CD4 lymphocyte count; Cross-sectional studies; Acquired immunodeficiency syndrome.

INTRODUCTION:

Antiretroviral therapy has converted infection with the human immunodeficiency virus from a rapidly fatal illness into a chronic condition, and the causes of death and disability in this population have shifted accordingly towards non-communicable disease.¹ Cardiovascular disease is prominent among these: a systematic review of longitudinal studies estimated that infection roughly doubles the risk of cardiovascular disease, and the share of the global

cardiovascular burden attributable to it more than doubled over the twenty-six years to 2015.² Cardiac involvement is not a new observation. Two-dimensional echocardiography identified dilated cardiomyopathy, pericardial effusion and other abnormalities in patients studied in the earliest years of the epidemic,³ and the recognised spectrum has since widened to encompass myocardial, pericardial, valvular and vascular disease.⁴



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Dilated cardiomyopathy is the classical myocardial phenotype. Defined by left ventricular dilatation with impaired systolic function in the absence of abnormal loading conditions or coronary disease sufficient to account for the finding,⁵ it has been reported far more often in this population than in the general population, and a meta-analysis conducted in the era of highly active antiretroviral therapy found systolic and diastolic impairment to be common even among patients with few or no symptoms.⁶ Valvular disease has attracted less attention. In a large South African registry, concurrent infection accounted for almost one in ten de novo presentations of heart disease in a setting where rheumatic valve disease remains endemic,⁷ and infective endocarditis constitutes a further distinct mechanism of valve injury in this population.⁸

The mechanisms are multiple and overlapping. Direct myocardial infection, cytokine-mediated injury, opportunistic co-infection, nutritional deficiency and autoimmune responses have all been implicated in the pathogenesis of myocardial disease,⁹ and the resulting cardiomyopathy has a clinical profile of its own.¹⁰ Antiretroviral therapy contributes independently through dyslipidaemia, insulin resistance and mitochondrial toxicity, particularly with protease inhibitors and thymidine-analogue nucleoside reverse transcriptase inhibitors.¹¹ Left ventricular dysfunction in this setting is not benign, having been associated with an increased risk of sudden cardiac death.¹²

Reported prevalence varies widely with setting and with the diagnostic criteria applied. In a large multisite cohort in the treatment era, echocardiographic abnormalities were common despite generally well-controlled infection,¹³ and diastolic dysfunction together with increased left ventricular mass was substantially more frequent than in uninfected comparators.¹⁴ African series report a comparable burden: structural echocardiographic abnormality in a Nigerian cohort proved independent of CD4 count,¹⁵ and cross-sectional studies from Lagos¹⁶ and Yaoundé¹⁷ documented frequent echocardiographic and electrocardiographic abnormality, although the determinants of cardiac disease in these populations remain incompletely defined.¹⁸

Nepal has a concentrated epidemic sustained among key populations, with an adult prevalence of about 0.2% that has declined progressively, and an expanding national treatment programme that has increased the number of people surviving with long-standing infection.¹⁹ Despite this growing treated population, the burden of structural cardiac disease in Nepal has not been characterised, and no local estimate exists to guide whether echocardiography should form part of routine care. The present study was undertaken to address that gap.

Objectives:

The primary objective was to estimate, over the twelve-month period from August 2018 to July 2019, the prevalence of structural heart disease, defined as dilated cardiomyopathy or valvular heart disease on transthoracic echocardiography, among adults receiving antiretroviral therapy at a tertiary referral centre in eastern Nepal. The secondary objective was to determine, in the same cohort, whether the presence of structural heart disease and of each of its two component lesions was associated with age, sex or CD4 count.

MATERIALS AND METHODS:

Study design and setting:

This was a hospital-based, cross-sectional study conducted in the Department of Internal Medicine of B.P. Koirala Institute of Health Sciences, Dharan, a tertiary referral centre serving eastern Nepal. Data were collected over a twelve-month period, from August 2018 to July 2019, in the medical wards, outpatient department and other inpatient units of the institute.

Study population and eligibility:

Adults with serologically confirmed infection with the human immunodeficiency virus who were established on antiretroviral therapy were eligible. To isolate cardiac abnormality attributable to the infection or its treatment from that attributable to conventional causes, patients were excluded if they had exposure to any drug with recognised cardiotoxic action; a history of alcohol dependence, defined according to Harrison's criteria as repeated alcohol-related difficulties in at least two of eleven life areas clustering within the same twelve-month period; a documented history of heart disease including coronary artery disease, arrhythmia, congenital heart disease or angina; systemic arterial hypertension; or a systemic disorder such as malnutrition, diabetes mellitus, or rheumatic, neurological or neoplastic disease.

Sampling:

Consecutive sampling was used, and every eligible patient encountered during the study period who provided written informed consent was enrolled. A total of 126 participants was recruited. No a priori sample size calculation was performed; the achieved sample was determined by the number of eligible patients presenting within the fixed study period.

Data collection and measurements:

A predesigned, pretested questionnaire was administered to record demographic details comprising age, sex, address and occupation. Literacy was classified using the definition of the United Nations Educational, Scientific and Cultural Organization, namely the ability to identify, understand, interpret, create, communicate and compute using printed and written materials associated with varying contexts.

Socioeconomic status was assigned using the modified Kuppuswamy scale,²⁰ which combines education and occupation of the head of the household with total monthly family income; for analysis the standard strata were collapsed into lower and middle categories, as no participant reached the upper stratum.

All participants underwent haemoglobin and haematocrit estimation, total leucocyte and platelet counts, blood urea, serum creatinine, liver function tests and CD4 lymphocyte enumeration. Transthoracic two-dimensional and Doppler echocardiography was then performed by the same operator to identify structural abnormality of the heart.

Definitions of outcomes:

Structural heart disease was used as an umbrella term for conditions arising from defects or abnormalities of the cardiac valves, walls and muscle, and for the purposes of this study comprised dilated cardiomyopathy, valvular heart disease, or both. Dilated cardiomyopathy was diagnosed when diffuse left ventricular hypokinesia with an ejection fraction below 45% was accompanied by left ventricular dilatation, taken as a left ventricular end-diastolic volume index exceeding 80 mL/m², a definition consistent with the phenotypic description of dilated cardiomyopathy in international classification.⁵ Valvular heart disease was diagnosed after evaluation of all components of the valve apparatus, including the leaflets, annulus, chordae tendineae and papillary muscles, for prolapse, flail or thickened leaflets, retraction or tethering, perforation, vegetation, chordal elongation or rupture, papillary muscle rupture, and commissural fusion.

Statistical analysis:

Data were entered in Microsoft Excel and analysed using the Statistical Package for the Social Sciences (SPSS). Categorical

variables are summarised as frequency and percentage in the format n (%), and continuous variables as mean $\pm$ standard deviation. Prevalence estimates are accompanied by 95% confidence intervals calculated by the Wilson score method. Differences in categorical variables between groups were tested using Pearson's chi-square test, with Fisher's exact test substituted where any expected cell count was below five; the strength of association is additionally expressed as an odds ratio with a 95% confidence interval derived by the Woolf method. Continuous variables were compared using the independent-samples t test, and both the test statistic with its degrees of freedom and the corresponding p value are reported. A two-sided p value below 0.05 was taken as statistically significant.

RESULTS:

A total of 126 patients were enrolled, and the sociodemographic and immunological profile of the cohort is shown in Table 1. Fifty-one participants (40.5%) were aged 31 to 40 years, 46 (36.5%) were aged 41 years or more and 29 (23.0%) were 30 years or younger, with a mean age of 37 ± 10 years. The sexes were almost evenly represented, with 64 men (50.8%) and 62 women (49.2%). One hundred and eleven participants (88.1%) were literate and 15 (11.9%) were illiterate, while 66 (52.4%) were employed and 60 (47.6%) were unemployed. The cohort was predominantly Hindu, comprising 123 participants (97.6%), with 1 Muslim participant (0.8%) and 2 (1.6%) of other faiths. Seventy-one participants (56.3%) belonged to the lower and 55 (43.7%) to the middle socioeconomic stratum. Immunological reconstitution was generally good: the mean CD4 count was 563 ± 260 cells/ μ L, and only 6 participants (4.8%) had a count below 200 cells/ μ L, the remaining 120 (95.2%) having counts of 200 cells/ μ L or above.

Table 1: Sociodemographic and immunological characteristics of the study participants (n = 126)

Characteristic	n (%)
Age group (years)	
≤30	29 (23.0)
31–40	51 (40.5)
≥41	46 (36.5)
Mean ± SD	37 ± 10
Sex	
Male	64 (50.8)
Female	62 (49.2)
Literacy	
Literate	111 (88.1)
Illiterate	15 (11.9)
Occupation	
Employed	66 (52.4)
Unemployed	60 (47.6)
Religion	
Hindu	123 (97.6)
Muslim	1 (0.8)
Other	2 (1.6)
Socioeconomic status	
Lower	71 (56.3)
Middle	55 (43.7)
CD4 count (cells/μL)	
<200	6 (4.8)
≥200	120 (95.2)
Mean ± SD	563 ± 260

SD, standard deviation.

Structural heart disease was identified in 19 participants, giving a prevalence of 15.1% (95% confidence interval 9.9–22.4), while 107 participants (84.9%) had no structural abnormality (Table 2). Dilated cardiomyopathy was the commoner of the two component lesions and was present in 14 participants (11.1%, 95% confidence interval 6.7–17.8), whereas valvular heart disease was found in 7 (5.6%, 95% confidence interval 2.7–11.0). Because the two lesions coexisted in 2 participants (1.6%), the number of patients with any structural abnormality was 19 rather than 21.

Table 2: Prevalence of structural heart disease on echocardiography (n = 126)

Echocardiographic finding	n (%)	95% CI (%)
Structural heart disease (any)	19 (15.1)	9.9–22.4
Dilated cardiomyopathy	14 (11.1)	6.7–17.8
Valvular heart disease	7 (5.6)	2.7–11.0
Both lesions in the same patient	2 (1.6)	0.4–5.6
No structural heart disease	107 (84.9)	77.6–90.1

CI, confidence interval. Confidence intervals calculated by the Wilson score method.

The distribution of structural heart disease across demographic and immunological strata is presented in Table 3. Among the 19 affected participants, 6 (31.6%) were 30 years of age or younger, 7 (36.8%) were aged 31 to 40 years and 6 (31.6%) were 41 years or older, a distribution that did not differ significantly from that of unaffected participants ($\chi^2 = 0.94$, $df = 2$, $p = 0.627$). Mean age was likewise comparable between those with and without structural heart disease, at 38 ± 10 and 37 ± 10 years respectively ($t = 0.40$, $df = 124$, $p = 0.689$). Structural heart disease was present in 11 of 64 men (17.2%) and 8 of 62 women (12.9%), a difference that was not statistically significant ($\chi^2 = 0.45$, $df = 1$, $p = 0.502$; odds ratio 1.40, 95% confidence interval 0.52–3.76). Mean CD4 count was numerically higher in participants with structural heart disease than in those without, at 627 ± 309 compared with 551 ± 248 cells/ μ L, but the difference was not statistically significant ($t = 1.18$, $df = 124$, $p = 0.239$).

Table 3: Association of structural heart disease with age, sex and CD4 count

Variable	Present (n=19)	Absent (n=107)	Test statistic	p value
Age group (years)			$\chi^2 = 0.94$, $df = 2$	0.627
≤30	6 (31.6)	23 (21.5)		
31–40	7 (36.8)	44 (41.1)		
≥41	6 (31.6)	40 (37.4)		
Age, years (mean ± SD)	38 ± 10	37 ± 10	$t = 0.40$, $df = 124$	0.689
Sex			$\chi^2 = 0.45$, $df = 1$	0.502
Male	11 (57.9)	53 (49.5)		
Female	8 (42.1)	54 (50.5)		
CD4 count, cells/ μ L (mean ± SD)	627 ± 309	551 ± 248	$t = 1.18$, $df = 124$	0.239

Values are n (%) unless otherwise stated. χ^2 , Pearson's chi-square test; df , degrees of freedom; SD, standard deviation. Odds ratio for male sex 1.40 (95% confidence interval 0.52–3.76).

Analysis restricted to dilated cardiomyopathy produced the same pattern (Table 4). Participants with dilated cardiomyopathy did not differ in age from those without, with means of 39 ± 10 and 37 ± 10 years ($t = 0.71$, $df = 124$, $p = 0.482$). The lesion was present in 8 of 64 men (12.5%) and 6 of 62 women (9.7%), which was not a significant difference ($\chi^2 = 0.25$, $df = 1$, $p = 0.614$; odds ratio 1.33, 95% confidence interval 0.43–4.09). Mean CD4 count was 667 ± 318 cells/ μ L in those with dilated cardiomyopathy and 550 ± 247 cells/ μ L in those without; although the difference of 117 cells/ μ L was the largest observed in the study, it did not reach statistical significance ($t = 1.62$, $df = 124$, $p = 0.109$).

Table 4: Association of dilated cardiomyopathy with age, sex and CD4 count

Variable	Present (n=14)	Absent (n=112)	Test statistic	p value
Age, years (mean ± SD)	39 ± 10	37 ± 10	$t = 0.71$, $df = 124$	0.482
Sex			$\chi^2 = 0.25$, $df = 1$	0.614
Male	8 (57.1)	56 (50.0)		
Female	6 (42.9)	56 (50.0)		
CD4 count, cells/ μ L (mean ± SD)	667 ± 318	550 ± 247	$t = 1.62$, $df = 124$	0.109

Values are n (%) unless otherwise stated. χ^2 , Pearson's chi-square test; df , degrees of freedom; SD, standard deviation. Odds ratio for male sex 1.33 (95% confidence interval 0.43–4.09).

Valvular heart disease showed no association with any of the variables examined (Table 5). The 7 affected participants were slightly younger than the 119 unaffected participants, with means of 34 ± 7 and 37 ± 10 years, but this difference was not significant ($t = -0.78$, $df = 124$, $p = 0.436$). Five of 64 men (7.8%) and 2 of 62 women (3.2%) were affected, giving an odds ratio

of 2.54 (95% confidence interval 0.47–13.62) that did not reach significance on Fisher's exact test ($p = 0.440$). Mean CD4 counts were almost identical in the two groups, at 593 ± 273 and 561 ± 256 cells/ μ L ($t = 0.32$, $df = 124$, $p = 0.749$).

Table 5: Association of valvular heart disease with age, sex and CD4 count

Variable	Present (n=7)	Absent (n=119)	Test statistic	p value
Age, years (mean $\pm$ SD)	34 $\pm$ 7	37 $\pm$ 10	$t = -0.78$, $df = 124$	0.436
Sex			Fisher's exact test	0.440
Male	5 (71.4)	59 (49.6)		
Female	2 (28.6)	60 (50.4)		
CD4 count, cells/ μ L (mean $\pm$ SD)	593 $\pm$ 273	561 $\pm$ 256	$t = 0.32$, $df = 124$	0.749

Values are n (%) unless otherwise stated. df, degrees of freedom; SD, standard deviation. Fisher's exact test used because an expected cell count was below five. Odds ratio for male sex 2.54 (95% confidence interval 0.47–13.62).

DISCUSSION:

In this cohort of 126 adults receiving antiretroviral therapy in eastern Nepal, structural heart disease was present in 19 participants (15.1%), with dilated cardiomyopathy in 14 (11.1%) and valvular heart disease in 7 (5.6%). None of the abnormalities had been suspected clinically before echocardiography, since patients with a known cardiac history were excluded at recruitment. The central observation is therefore that approximately one in seven treated adults with no cardiac history and largely reconstituted immunity harboured a structural cardiac lesion.

The prevalence of dilated cardiomyopathy observed here, 11.1%, is close to the figure of 11% reported in one of the earliest echocardiographic surveys of the disease, conducted before effective therapy was available.³ That correspondence is unexpected, because our participants were established on treatment and largely immunologically reconstituted, and series from the treatment era generally report less overt systolic dysfunction and a shift towards diastolic abnormality.^{13,14} The most plausible explanation lies in the diagnostic threshold applied rather than in a genuinely greater burden of myocardial disease. This study defined dilated cardiomyopathy by a single functional and dimensional criterion, an ejection fraction below 45% together with a raised end-diastolic volume index, whereas investigators who additionally require a fractional shortening below 28% or a specified end-diastolic diameter will classify fewer patients as affected. Part of what is labelled dilated cardiomyopathy in the present cohort may therefore represent milder systolic impairment that others would have categorised separately, a distinction that matters because subclinical impairment is now recognised as the dominant pattern in treated patients.⁶ Conversely, because diastolic function was not assessed, the

total burden of cardiac abnormality was almost certainly underestimated: where a comprehensive protocol has been applied, diastolic dysfunction has emerged as the commonest abnormality.¹⁴

The prevalence of valvular heart disease, at 5.6%, is difficult to attribute to the infection itself in a setting where rheumatic fever remains endemic. Registry data from sub-Saharan Africa show that concurrent infection contributes materially to de novo presentations of heart disease in populations carrying a high background burden of rheumatic valve disease,⁷ while infective endocarditis represents a separate and distinguishable mechanism of valve injury.⁸ Rheumatic valvulitis, degenerative change and infection-associated valve disease cannot be separated on the basis of the echocardiographic descriptors recorded here, and the absence of a virus-negative comparison group prevents any inference about excess risk. The most defensible interpretation is that valvular lesions occurred in this cohort at a rate broadly consistent with the regional background, and that their detection matters clinically irrespective of aetiology.

No association was found between structural heart disease and age or sex, whether the outcome was considered as a whole or separated into its component lesions. This is consistent with the view that myocardial involvement in this setting is driven by disease-specific processes such as cytokine-mediated injury and drug toxicity rather than by conventional cardiovascular risk profiles,^{9,11} and with reports that structural echocardiographic abnormality is not predicted by conventional markers of disease severity.¹⁵ The exclusion of hypertensive, diabetic and alcohol-dependent patients from the present cohort would have further attenuated any age-related

gradient. The male preponderance suggested by the odds ratios for each lesion was consistent in direction but imprecise, with confidence intervals spanning unity in every comparison, and cannot be interpreted as evidence of a sex difference.

The relationship with CD4 count merits particular comment because the direction of the observed difference is counter-intuitive. Participants with structural heart disease had a numerically higher mean CD4 count than those without, and the same pattern was seen for dilated cardiomyopathy, although neither difference reached statistical significance. The published evidence is not uniform. Earlier work linked advanced immunosuppression to pericardial and myocardial disease,^{3,4} whereas a Nigerian cohort applying a comparable structural definition found echocardiographic abnormality to be independent of CD4 count,¹⁵ and analyses of determinants in similar African populations have likewise failed to establish a consistent gradient.¹⁸ Two features of the present cohort make it particularly ill-suited to detecting one. First, immunosuppression was rare, with only 6 participants (4.8%) having counts below 200 cells/ μ L, so the range across which any CD4 gradient could express itself was largely absent. Second, a single cross-sectional measurement made after a period of treatment reflects immune reconstitution rather than the nadir, and it is the nadir that has been shown to correlate with left ventricular mass and diastolic dysfunction.¹⁴ The reasonable conclusion is that these data are uninformative about the CD4 relationship, not that a protective effect of immunosuppression exists.

These findings carry a practical implication. Because the abnormalities detected were clinically silent and unrelated to the demographic and immunological variables routinely recorded in treatment programmes, there is no subgroup within a treated population that could be selected for targeted investigation on the basis of age, sex or current CD4 count. If structural cardiac disease is to be identified before it becomes symptomatic, screening must be applied to the treated population as a whole, and echocardiography, being non-invasive, radiation-free and increasingly available at district level, is the natural modality. The recognition that treatment itself introduces metabolic and body-composition changes that raise cardiovascular risk over time¹¹ reinforces the case for integrating cardiac assessment into routine care rather than reserving it for those who present with symptoms.

Several limitations qualify these conclusions. The cross-sectional design establishes prevalence but cannot address incidence, causality or the temporal relationship between infection, treatment and cardiac abnormality. Recruitment from a single tertiary referral centre by consecutive sampling, without a formal sample size calculation, limits generalisability to the wider Nepalese population and leaves

the prevalence estimates imprecise, as reflected in the width of the confidence intervals; the small numbers of participants with each lesion, particularly the 7 with valvular heart disease, meant that the study was substantially underpowered to detect associations of the magnitude reported in larger cohorts, and the null findings should not be read as evidence of absence. The absence of a virus-negative control group means that no abnormality can be attributed to the infection or its treatment rather than to background population risk. Duration of infection, duration and class of antiretroviral therapy, nadir CD4 count and plasma viral load were not recorded, which removes the exposure variables most likely to explain myocardial involvement and prevents any assessment of drug-specific cardiotoxicity. The echocardiographic protocol assessed systolic function and valve structure but not diastolic function, chamber quantification indexed to body surface area, pericardial effusion, pulmonary artery pressure or myocardial strain, so the total burden of cardiac abnormality was almost certainly underestimated; the diagnostic thresholds used for dilated cardiomyopathy also differ from those applied in several of the comparator studies, which constrains direct comparison. Studies were performed by a single operator without a second independent reader, so inter-observer reproducibility could not be assessed. Finally, socioeconomic strata were collapsed into two categories, and this variable was not carried into the analysis of outcomes.

CONCLUSION:

Structural heart disease was present in 19 of 126 adults (15.1%) receiving antiretroviral therapy, comprising dilated cardiomyopathy in 14 (11.1%) and valvular heart disease in 7 (5.6%), and was not associated with age, sex or CD4 count. Because these lesions were clinically silent and could not be predicted from routinely collected variables, selective investigation is unlikely to identify them. Echocardiographic screening of treated adults, irrespective of symptoms or immunological status, is therefore recommended. Larger multicentre studies incorporating a virus-negative comparison group, diastolic and strain assessment, and treatment exposure data are needed to define the attributable burden and its determinants.

DECLARATIONS:

Ethical Approval: The study protocol was approved by the Institutional Review Committee of B.P. Koirala Institute of Health Sciences (reference number 499/075/076-IRC, dated 29 July 2018). Written informed consent was obtained from every participant before enrolment, after the purpose, procedures and voluntary nature of participation had been explained in a language the participant understood. No identifying information was recorded in the study database.

Informed Consent: Informed consent was obtained from all participants prior to their inclusion in the study. Participants were informed that their data would be kept confidential and used solely for research purposes.

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Conflict of Interest: The authors declare that there is no conflict of interest.

Data availability statement: The dataset presented in this study is available on request from the corresponding author.

AI Use Disclosure: No generative AI tools were used in the writing or data analysis. AI tools were limited to language assistance. The authors remain fully responsible for the content.

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REFERENCES:

1. Croxford S, Kitching A, Desai S, Kall M, Edelstein M, Skingsley A, et al. Mortality and causes of death in people diagnosed with HIV in the era of highly active antiretroviral therapy compared with the general population: an analysis of a national observational cohort. *Lancet Public Health*. 2017;2(1):e35-46. [https://doi.org/10.1016/S2468-2667\(16\)30020-2](https://doi.org/10.1016/S2468-2667(16)30020-2)
2. Shah ASV, Stelzle D, Lee KK, Beck EJ, Alam S, Clifford S, et al. Global burden of atherosclerotic cardiovascular disease in people living with HIV: systematic review and meta-analysis. *Circulation*. 2018;138(11):1100-12. <https://doi.org/10.1161/CIRCULATIONAHA.117.033369>
3. Himelman RB, Chung WS, Chernoff DN, Schiller NB, Hollander H. Cardiac manifestations of human immunodeficiency virus infection: a two-dimensional echocardiographic study. *J Am Coll Cardiol*. 1989;13(5):1030-6. [https://doi.org/10.1016/0735-1097\(89\)90256-8](https://doi.org/10.1016/0735-1097(89)90256-8)
4. Yunis NA, Stone VE. Cardiac manifestations of HIV/AIDS: a review of disease spectrum and clinical management. *J Acquir Immune Defic Syndr Hum Retrovirol*. 1998;18(2):145-54. <https://doi.org/10.1097/00042560-199806010-00006>
5. Elliott P, Andersson B, Arbustini E, Bilinska Z, Cecchi F, Charron P, et al. Classification of the cardiomyopathies: a position statement from the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J*. 2008;29(2):270-6. <https://doi.org/10.1093/eurheartj/ehm342>
6. Cerrato E, D'Ascenzo F, Biondi-Zoccai G, Calcagno A, Frea S, Grosso Marra W, et al. Cardiac dysfunction in pauci symptomatic human immunodeficiency virus patients: a meta-analysis in the highly active antiretroviral therapy era. *Eur Heart J*. 2013;34(19):1432-6. <https://doi.org/10.1093/eurheartj/ehs471>
7. Sliwa K, Carrington MJ, Becker A, Thienemann F, Ntsekhe M, Stewart S. Contribution of the human immunodeficiency virus/acquired immunodeficiency syndrome epidemic to de novo presentations of heart disease in the Heart of Soweto Study cohort. *Eur Heart J*. 2012;33(7):866-74. <https://doi.org/10.1093/eurheartj/ehr398>
8. Nel SH, Naidoo DP. An echocardiographic study of infective endocarditis, with special reference to patients with HIV. *Cardiovasc J Afr*. 2014;25(2):50-7. <https://doi.org/10.5830/CVJA-2013-084>
9. Barbaro G. Pathogenesis of HIV-associated heart disease. *AIDS*. 2003;17 Suppl 1:S12-20. <https://doi.org/10.1097/00002030-200304001-00003>
10. Barbaro G. HIV-associated cardiomyopathy: etiopathogenesis and clinical aspects. *Herz*. 2005;30(6):486-92. <https://doi.org/10.1007/s00059-005-2728-z>
11. Grinspoon S, Carr A. Cardiovascular risk and body-fat abnormalities in HIV-infected adults. *N Engl J Med*. 2005;352(1):48-62. <https://doi.org/10.1056/NEJMra041811>
12. Moyers BS, Secemsky EA, Vittinghoff E, Wong JK, Havlir DV, Hsue PY, et al. Effect of left ventricular dysfunction and viral load on risk of sudden cardiac death in patients with human immunodeficiency virus. *Am J Cardiol*. 2014;113(7):1260-5. <https://doi.org/10.1016/j.amjcard.2013.12.036>
13. Mondy KE, Gottdiener J, Overton ET, Henry K, Bush T, Conley L, et al. High prevalence of echocardiographic abnormalities among HIV-infected persons in the era of highly active antiretroviral therapy. *Clin Infect Dis*. 2011;52(3):378-86. <https://doi.org/10.1093/cid/ciq066>
14. Hsue PY, Hunt PW, Ho JE, Farah HH, Schnell A, Hoh R, et al. Impact of HIV infection on diastolic function and left ventricular mass. *Circ Heart Fail*.

- 2010;3(1):132-9.
<https://doi.org/10.1161/CIRCHEARTFAILURE.109.854943>
15. Ogunmodede JA, Kolo PM, Katibi IA, Salami AK, Omotoso ABO. Structural echocardiographic abnormalities seen in HIV/AIDS patients are independent of CD4 count. Niger J Clin Pract. 2017;20(6):716-23. <https://doi.org/10.4103/1119-3077.208954>
16. Olusegun-Joseph DA, Ajuluchukwu JN, Okany CC, Mbakwem AC. Echocardiographic patterns in treatment-naïve HIV-positive patients in Lagos, south-west Nigeria. Cardiovasc J Afr. 2012;23(8):e1-6. <https://doi.org/10.5830/CVJA-2012-048>
17. Hamadou B, Ngweth MN, Fotso MM, Mfeukeu-Kuate L, Jingi AM, Noubiap JJ, et al. Echocardiographic and electrocardiographic abnormalities in adults living with human immunodeficiency virus: a cross-sectional study in the Yaoundé Central Hospital, Cameroon. Cardiovasc Diagn Ther. 2017;7(6):607-15. <https://doi.org/10.21037/cdt.2017.10.12>
18. Isiguzo G, Okeahialam B, Danbauchi S, Odili A, Iroezindu M. Determinants of HIV-related cardiac disease among adults in north central Nigeria. Heart Asia. 2013;5(1):130-5. <https://doi.org/10.1136/heartasia-2013-010284>
19. Paudel T, Singh N, Banjara MR, Kafle SP, Ghimire YC, Pokharel BR, et al. Epidemiology of HIV, programmatic progress and gaps in last 10 years in Nepal. J Virus Erad. 2016;2(Suppl 4):35-40. [https://doi.org/10.1016/S2055-6640\(20\)31099-2](https://doi.org/10.1016/S2055-6640(20)31099-2)
20. Sharma R. Revised Kuppaswamy's socioeconomic status scale: explained and updated. Indian Pediatr. 2017;54(10):867-70. <https://doi.org/10.1007/s13312-017-1151-x>