Original Article

Left ventricular mass and diastolic dysfunction in children infected with the human immunodeficiency virus

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ABSTRACT

Background: Increased left ventricular mass (LVM) and diastolic dysfunction are associated with higher morbidity and mortality among adult patients with human immunodeficiency virus (HIV) infection.

Objective: The objective of the following study is to determine the prevalence of increased LVM and diastolic dysfunction in Nigerian children infected with HIV.

Methods: Cross-sectional comparative study of LVM and left ventricular (LV) diastolic function of 150 HIV-positive children and controls asymptomatic for cardiac disease.

Results: Mean LVM was larger in subjects than controls - 66.5 (95% confidence interval, 63.7-69.3) versus 56.9 (54.1-59.7) g/m^2 respectively - $P < 0.001$. An increased LVM was present in 21 (14.0%) subjects and 4 (2.7%) controls - $P < 0.001$. Mean mitral valve peak flow velocities and pressure gradients for the early and late diastolic waves were higher among HIV positive children than controls ($P < 0.001$). LV diastolic dysfunction was present in 46 (30.7%) subjects and 19 (12.7%) controls ($P < 0.001$). Subjects with increased LVM were younger and had more severe disease than those with normal LVM. Subjects and controls were similar with respect to their clinical and immunological stages of disease and use of nucleoside reverse transcriptase inhibitors.

Conclusion: Increased LVM and diastolic dysfunction are significantly more common in HIV-infected children compared with controls and occur in asymptomatic subjects.

KEY WORDS: Children, diastolic dysfunction, human immunodeficiency virus, left ventricular mass

INTRODUCTION

There have been several reports on the spectrum of cardiovascular complications of the human immunodeficiency virus (HIV) infection in adults but fewer in children.[1-3] Many of these studies have found HIV infection to be associated with a higher prevalence of cardiomyopathy and increased left ventricular mass (LVM).[4-6] An increase in LVM may be associated with left ventricular (LV) diastolic dysfunction and both have been shown to increase morbidity and mortality in both adults and children, with increased predisposition to ventricular arrhythmias, severe heart failure, myocardial infarction and cerebrovascular accidents.[7-9]

The pathogenesis of increased LVM which often underlies diastolic dysfunction, may be related to an increase in sympathetic tone, a manifestation of autonomic dysfunction, which in turn raises the concentration of catecholamines, leading eventually to myocardial hypertrophy.[10,11] HIV infected adults receiving nucleoside reverse transcriptase inhibitors (NRTI) have been found to have higher LVM compared with controls.[12] Higher LVM has also been shown to be more common in older HIV-infected children and those with opportunistic infections and malnutrition.[13,14]

The cause of LV diastolic dysfunction in HIV infected individuals is still uncertain, but possible etiologic factors have been proposed. Higher levels of inflammatory markers such as high sensitivity C-reactive protein have been linked to LV diastolic dysfunction in the general population.[15] In HIV infected individuals, the HIV virions and other viruses infect the myocardial cells directly, leading to the local release of cytokines. Cytokines act by down-regulating the sarcoendoplasmic reticulum Ca^{2+}-ATPase pumps which results in a slower removal of cytosolic calcium. This could lead to a prolonged and incomplete diastolic phase and consequently, diastolic dysfunction.[16,17] Hsue *et al.*[10] in their study have

reported that the use of NRTI and lower CD4 counts were associated with LV diastolic dysfunction, while a decrease in CD4+ T cell count of <100 cell/mm^3 resulted in an increase in the mean LVM by 1.3 g.

LV diastolic dysfunction is known to precede systolic dysfunction in most cardiac diseases and has also been reported in HIV-infected children.[4,18-20] Very few studies on the myocardial function of such children have emanated from sub-Saharan Africa, where the greatest burden of the disease lies and none of these has focused on diastolic function.[21,22] Okoromah *et al*.[21] in South-west Nigeria showed that the mean LVM was significantly higher in HIV-infected children compared with controls, but did not assess their diastolic function. The current report aims to determine the prevalence of increased LVM and diastolic dysfunction in Nigerian African children infected with HIV and their possible associated factors.

MATERIALS AND METHODS

A total of 150 children with confirmed HIV infection attending the pediatric infectious disease clinic (PIDC) of the Jos University Teaching Hospital (JUTH) were systematically sampled and enrolled into the study. Age-and sex-matched HIV-negative children of the same number, with no acute or chronic illnesses were recruited from the pediatric out-patients department and the PIDC and constituted the control group. Socio-economic status of all the participants was determined according to the method described by Olusanya *et al*.[23]

Written informed consent was obtained from each child's parent or guardian, while approval was obtained from the Ethics Committee of JUTH before commencement of the study.

Two-dimensional, M-mode and Doppler echocardiograms were performed on all subjects and controls using an Acuson® Cypress® Ultrasound System (Siemens Medical, Germany, 2004) with 3.5 MHz and 7.5 MHz transducers for older and younger children respectively. Our observations on the LV systolic function of the study population have been previously reported.[22]

End-diastolic measurements of the left ventricular internal dimension (LVID), interventricular septal thickness (IVST) and posterior wall thickness (PWT) – all measured according to the guidelines of the American Society of Echocardiography[24] were used to calculate LV mass using the formula:

Anatomic LVM = 1.04 ([LVID + PWT + IVST]^3 – [LVID]^3) – 13.6 g.[25]

LVM was indexed to the body surface area in m^2, compared with published normal values for age and adjudged to be increased if above the normal range for age.[26]

LV diastolic function was assessed using pulsed wave Doppler flow velocities across the mitral valve. The peak velocity (V_{max}) and peak pressure gradient (ΔP_{max}) across the mitral valve in early and late diastole corresponding to the early (E) and atrial (A) waves were measured and the E/A ratio calculated. LV diastolic dysfunction was adjudged present if the mitral valve E/A wave ratio was <1 (slow-relaxation pattern) or above 2.5 (restrictive pattern).[27]

RESULTS

The children were aged between 7 months and 14 years with a mean of 5.59 (5.2-6.0) years for the HIV positive subjects and 5.6 (5.0-6.2) years for the controls and a male to female ratio of 1.1:1. Subjects differed from the controls with respect to their socio-economic status [Table 1]. The most likely route of acquisition of the HIV infection was maternal to child (vertical) transmission in the majority (95.3%) of cases, followed by blood transfusion in 6 (4.0%). In 1 (0.7%) patient, the route of transmission could not be ascertained. This was a 13-year-old boy whose mother was HIV negative and who had no history of blood transfusion, sexual contact, traditional circumcision, uvulectomy or scarification marks. Features suggestive of encephalopathy were present in only one patient [Table 1].

LV mass

The mean LVM was 66.5 (63.7-69.3) g/m^2 in HIV-infected subjects compared with 56.9 (54.1-59.7) g/m^2 in HIV negative controls. The difference was statistically significant - $P < 0.001$ [Table 2]. Excessive LVM was

Table 1: Clinical characteristics of HIV positive subjects, age and socio-economic status distribution of subjects and controls

Characteristics	HIV positive (n=150) n (%)	HIV negative (n=150) n (%)	Chi-square	P value
Clinical characteristics				
Most likely route of transmission	-			
Vertical	143 (95.3)			
Blood transfusion	6 (4.0)			
Uncertain	1 (0.7)			
Encephalopathy	1 (0.7)	0 (0.0)	-	-
Age group (years)				
0-<1	2 (1.3)	2 (1.3)	0.02	1.00
1-<3	28 (18.7)	28 (18.7)		
3-<5	51 (34.0)	50 (33.3)		
5-<10	51 (34.0)	52 (34.7)		
10-<15	18 (12.0)	18 (12.0)		
Socio-economic status				
Upper	41 (27.3)	64 (42.7)	9.29	0.01*
Middle	45 (30.0)	28 (18.7)		
Lower	64 (42.7)	58 (38.6)		

*Statistically significant. HIV – Human immunodeficiency virus

present in 21 (14.0%) subjects compared with 4 (2.7%) controls - $P < 0.001$. Subjects with increased LVM were younger and had more severe disease than those with a normal LVM. There was no significant difference between subjects with excessive LVM compared with subjects without, in terms of their immunological stages of disease and the use of NRTI [Table 3].

LV diastolic dysfunction

The mean mitral valve V_{max} and ΔP_{max} for the early diastolic waves (E waves) were significantly higher in HIV-infected children (91.1 [90.2-92.0] cm/s and 3.3 [3.2-3.4] mmHg) than in controls (85.4 [84.3-86.6] cm/s and 3.0 [2.9-3.1] mmHg) respectively - $P < 0.001$ in each case. In late diastole during active atrial contraction, the mean A wave V_{max} and ΔP_{max} were also significantly higher in HIV-infected subjects than in HIV-negative controls - $P < 0.001$ in each case [Table 2].

LV diastolic dysfunction (of the restrictive pattern in all cases) was present in 46 (30.7%) subjects compared with 19 (12.7%) controls ($P < 0.001$). Although subjects with LV diastolic dysfunction were older than those without (6.0 [5.1-6.9] and 5.4 [5.1-5.7] years respectively), the difference was not statistically significant ($P = 0.36$). There was also no significant difference between subjects with LV diastolic dysfunction compared with subjects without the abnormality with respect to their clinical and immunological stages of disease and the use of NRTI [Table 4]. Multiple linear regression analysis revealed no significant correlation between diastolic dysfunction and factors such as tachycardia ($P = 0.41$), fever ($P = 0.71$), hypertension ($P = 0.30$) or hypotension ($P = 0.34$).

Table 2: Mean LVM and mitral valve flow parameters in HIV-positive subjects compared with HIV-negative controls

Parameters	Mean (95% CI)		*P* value
	HIV-positive (*n*=150)	HIV-negative (*n*=150)	
LVM (g/m²)	66.5 (65.1, 67.9)	56.9 (55.5, 57.3)	<0.001*
Mitral valve			
E wave			
V_{max} (cm/s)	91.1 (90.2, 92.0)	85.4 (84.3, 86.6)	<0.001*
ΔP_{max} (mmHg)	3.3 (3.2, 3.4)	3.0 (2.9, 3.1)	<0.001*
A wave			
V_{max} (cm/s)	58.6 (57.8, 59.4)	52.0 (51.0, 53.0)	<0.001*
ΔP_{max} (mmHg)	1.4 (1.36, 1.44)	1.1 (1.06, 1.14)	<0.001*
E/A ratio	1.71 (1.67, 1.73)	1.6 (1.58, 1.62)	0.04*

HIV – Human immunodeficiency virus; V_{max} – Peak velocity; ΔP_{max} – Peak pressure gradient; LVM – Left ventricular mass; E wave – Early wave; A wave – Atrial wave; CI – Confidence interval. *Statistically significant

Table 3: Increased LVM and the clinical characteristics of HIV positive subjects

Variable	Increased LVM (*n*=21)	Normal LVM (*n*=129)	*P* value
Age (years)			
Mean (95% CI)	3.9 (3.4, 4.1)	5.5 (5.2, 5.8)	0.002*
Sex (*n*)			
Male	12	61	0.4
Female	9	68	
Treatment with ART (*n*)			
Yes	10	74	0.4
No	11	55	
WHO clinical stage (*n*)			
I and II	17	94	0.77
III and IV	4	31	
WHO immunological stage (*n*)			
Not significant/mild	8	80	0.04*
Advanced/severe	13	49	

HIV – Human immunodeficiency virus; LVM – Left ventricular mass; CI – Confidence interval; ART – Antiretroviral therapy; WHO – World Health Organization. *Statistically significant

DISCUSSION

The impact of HIV infection on LV diastolic function has not been previously described in children in sub-Saharan Africa even though HIV is a common cause of morbidity and mortality in the region. Our study demonstrates that HIV infection is associated with both increased LV mass index and diastolic dysfunction.

We found a larger mean LVM among the HIV-positive children in this study compared with controls – similar to findings by others.[28,29] Immune activation in HIV-infected adults is postulated to contribute to the observed increase in LVM in a similar manner as with systemic lupus erythematosus, rheumatoid arthritis and other chronic inflammatory conditions in adults without HIV infection.[30,31] Our finding of a more advanced immunological stage of disease in those with larger muscle mass supports this hypothesis. In addition, tachycardia has been typically reported along with increased LVM in HIV-infected children, suggesting that the increased LVM may be as a result of autonomic dysfunction.[26] This has also been likened to the increase in heart muscle mass in response to the physical and metabolic stresses that are associated with chronic diseases and malnutrition.[32] We found no correlation between the presence of diastolic dysfunction and tachycardia in the HIV-infected children we studied. However, we did not further assess for autonomic dysfunction by evaluating heart rate variation during deep breathing and its response to postural changes, as has been done in adult subjects.[33]

Our study shows that LV diastolic dysfunction using pulse wave Doppler flow velocities across the mitral valve is significantly more common in HIV subjects compared with controls. Nearly 30.7% of our subjects had diastolic dysfunction, a figure similar to the 26% detected in adults by Mondy *et al.*[34] in the United States but higher than the 10% reported by Kumar *et al.*[35] in children. The

Table 4: Left ventricular diastolic dysfunction with respect to clinical characteristics of HIV-positive subjects

Variable	Diastolic dysfunction (*n*=46)	No diastolic dysfunction (*n*=104)	*P* value
Age (years)			
Mean (95% CI)	6.0 (5.1, 6.9)	5.4 (5.1, 5.7)	0.37
Sex (*n*)			
Male	25	55	0.87
Female	21	49	
Treatment with ART (*n*)			
Yes	19	66	0.23
No	27	38	
WHO clinical stage (*n*)			
I	21	67	0.69
II	10	11	
III	15	22	
IV	0	4	
WHO immunological stage (*n*)			
Not significant	17	47	0.73
Mild	8	16	
Advanced	6	15	
Severe	15	26	

ART – Antiretroviral therapy; WHO – World Health Organization; CI – Confidence interval; HIV – Human immunodeficiency virus

latter assessed LV diastolic dysfunction using the E/A ratio as in the present study, while Mondy *et al*.[33] in addition to the E/A ratio also assessed the mitral inflow deceleration time and the lateral mitral annular velocity using tissue Doppler. It is possible that we might have found a higher prevalence of LV diastolic dysfunction if we had also utilized these or other additional parameters.

HIV-associated cardiac dysfunction is said to be of multifactorial origin. The HIV or other associated viral infection may directly attack cardiac myocytes and thus impinge on myocardial function. The detection of HIV-1 in endomyocardial biopsy specimens of HIV patients with LV dysfunction and the detection of HIV-1 sequences in the myocytes of HIV patients undergoing autopsy provide some evidence for this.[36] Furthermore, ischemic heart disease may play a role – as suggested by the finding that HIV-positive adults have a demonstrable increase in their carotid artery intima-media thickness (IMT), which suggests subclinical atherosclerosis.[37] HIV-infected children have similarly been shown to have increased carotid artery IMT compared with healthy controls and this may also predispose them to diastolic dysfunction.[38]

We found no association between diastolic dysfunction in our HIV-infected children and the stage of their disease. Longo-Mbenza *et al*.[1] found a higher prevalence of diastolic dysfunction in adults with acquired immunodeficiency syndrome (stage IV disease). In contrast, only four children in our study had stage IV disease and none of them had diastolic dysfunction. However, we did not assess for the pseudo normalization pattern using mitral inflow deceleration time, which has been shown to correlate directly to LVM in adults.[39]

A longitudinal study design would have enabled us to monitor progression of LVM and LV diastolic dysfunction in our subjects and hence to determine the natural history of these conditions in children with HIV infection. The present study however has further shown the existence of these cardiac abnormalities in Nigerian children infected with HIV, thus strengthening the case for echocardiographic screening in these patients.

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