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Ventricular function and dimensions in children with human immunodeficiency virus infection

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Abstract

Background Prevalence of cardiac complications in children with human immunodeficiency virus (HIV) infection have increased, in association with the availability of antiretroviral (ARV) therapy and the decrease of opportunistic infections. However, studies on cardiac complications in HIV patients in the various HIV clinical and immunologic categories have been limited. Furthermore, cardiac complications in Indonesian HIV-infected children have never been reported.

Objectives To determine the prevalence of cardiac complications in HIV-infected children and to compare ventricular function and dimensions based on HIV clinical and immunologic categories.

Methods A cross-sectional study was done in the Department of Child Health, Cipto Mangunkusumo Hospital, Jakarta between October and December 2009 on 74 HIV-infected children aged below 15 years. Children with severe chronic or acute systemic diseases were excluded. Investigations included physical examinations, 12-lead electrocardiography and echocardiography to assess left ventricular dimension and ventricular function. Cardiac findings among children in different clinical and immunological categories were compared.

Results Five children showed left ventricular fractional shortening below 25% and 16 had right ventricular dysfunction. Mean cardiac function and dimension were in the normal range. No differences in cardiac function and dimension, among the clinical HIV category groups ($p>0.05$) or immunologic suppression status groups ($p>0.05$) were observed.

Conclusion In children with HIV infection, the prevalence of left ventricular dysfunction and right ventricular dysfunction was 7% and 22%, respectively. No differences in cardiac function and dimension were found among the different HIV clinical and immunological categories. **[Paediatr Indones. 2011;51:149-56]**.

Keywords: *HIV-infected children, ventricular function and dimension, clinical category*

Children infected with human immunodeficiency virus (HIV) may develop a wide range of asymptomatic and symptomatic cardiac abnormalities, some of which are known to be associated with poor survival. Several patterns of cardiovascular involvement have been reported, including asymptomatic left ventricle (LV) dysfunction, dilated cardiomyopathy, congestive heart failure, and hypotensive pump failure, all with cardiac-associated mortality.[1-3] These abnormalities occur frequently as the prevalence of HIV infection among children is high and advances in therapy and management improve life expectancy. Children with HIV who survive longer are less likely to die of pulmonary disease or infection, but more likely to die of cardiac causes.[4-6] The ability to identify patients at particularly high risk for cardiovascular abnormalities would improve survival and quality of life because care of HIV-infected patients relies on accurate diagnosis and prompt management of underlying illnesses, including symptomatic cardiac diseases.

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Subclinical cardiovascular abnormalities are common in HIV-infected children. The mean values for numerous cardiac echocardiographic parameters are significantly different from normal at baseline and may be used to predict adverse outcomes independently, as well as identify high-risk groups to target for early intervention and therapy. Echocardiographic abnormalities are common, persistent, and often progressive in children with HIV infection.[7] Studies reporting ventricular function and dimension according to clinical classification of HIV infection in children have been limited. Several risk factors for advanced cardiac involvement in HIV-infected children relating to HIV clinical category have been reported, including in those with HIV encephalopathy and low CD4 cell count, but not those who are asymptomatic or those with normal CD4 counts.[3,8,9] The purpose of this study was to determine the prevalence of cardiac abnormalities in children with HIV infection and to compare their ventricular function and dimensions according to HIV clinical and immunological categories.

Methods

A cross-sectional study was done in the Department of Child Health, Cipto Mangunkusumo Hospital, Jakarta between October and December 2009. Seventy-four consecutive HIV-infected children with positive HIV RNA or HIV antibodies on blood examination and aged under 15 years were included. Children with severe, acute or chronic disease such as pneumonia, thalassemia, sepsis, chronic renal failure, severe dehydration, severe malnutrition, shock, or those requiring mechanical ventilation were excluded. Clinical classification of HIV infection was defined at the time of first presentation and diagnosed according to the Clinical Categories by the Centers for Disease Control and Prevention (CDC) Revised Human Immunodeficiency Virus Pediatric Classification System.[10] Subjects underwent physical examination, 12-lead electrocardiography and echocardiography. The CD4 cell percentage data from within 4 weeks of subjects' enrollment were obtained. Subjects with CD4 $\geq$25% were considered as having no immunological suppression, while those with CD4 <25% were considered to be in an immunologically suppressed state. The study protocol was approved by the Ethics Committee of the Faculty of Medicine, University of Indonesia. Parental informed consent was obtained for each subject.

Comprehensive echocardiographic examination was done according to the American Society of Echocardiography Guidelines, including 2-D, M-Mode, pulsed, continuous, and color flow Doppler. Echocardiography was performed using a Phillips Agilent Sonos 4500 machine with 7 or 12 MHz sectoral transducer. Echocardiographic parameters were obtained three times, including function and dimension of left ventricle and right ventricular function by tricuspid annular plane systolic excursion (TAPSE). Two-dimensional echocardiography was used to assess the structural anatomy of the heart, including atrial situs, venoatrial connection, atrioventricular and ventriculoarterial connection, valves and pericardial effusion. M-mode was used to assess cardiac function and dimensions. Assessment was done simultaneously with ECG monitoring on cooperative or sleeping subjects. LV dimensions including LV mass, left ventricular end diastolic dimension (LVEDD), left ventricular end systolic dimension (LVESD), left ventricular posterior wall diastolic thickness (LVPWD), left ventricular posterior wall systolic thickness (LVPWS), interventricular septum diastolic thickness (IVSD), and interventricular septum systolic thickness (IVSS). LV systolic function was evaluated by measuring the ejection fraction (EF), fractional shortening (FS), cardiac output (CO), cardiac index (CI), and stroke volume (SV). LV diastolic function was evaluated by measuring the mitral valve E wave peak velocity, the mitral valve A wave peak velocity, deceleration time of the mitral valve, and TAPSE for RV function. Normal data based on previous studies was used as references.[2,7,11-13] Left ventricular dysfunction was defined as left ventricular fractional shortening (LVFS) below 25%, clinical congestive heart failure, previous receiving of cardiac medications, or abnormal echocardiographic parameters. Right ventricular dysfunction was defined as TAPSE of less than 15 mm or clinical evidence of right heart failure. Mild pericardial effusion was defined if the effusion was less than 5 mm in thickness, while effusion was considered

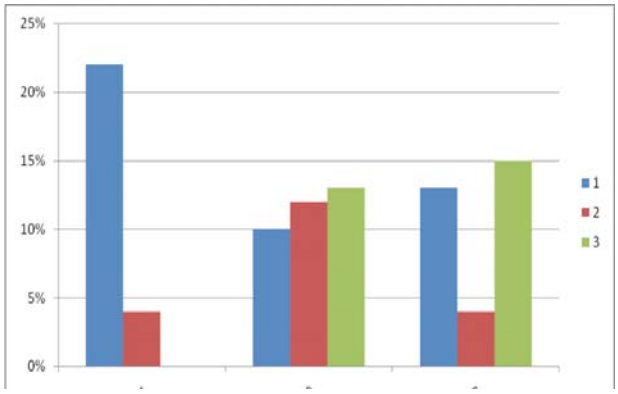
moderate to large if it was more than 5 mm thick on 2-D or M-Mode echocardiography.

Data analysis was performed using the SPSS 13.0 program. Data were expressed as mean (SD) or median and range depending on the normality test results. T-test and ANOVA were used to analyze the difference between ventricular function variables showing normal distribution, otherwise the data was analyzed using Mann-Whitney or Kruskal-Wallis test. A P value of less than 0.05 indicated statistical significance.

Results

During the study period, 82 children with HIV visited our ambulatory clinic, 74 of which were included in our study. Eight children were excluded because of severe pneumonia (3 children) or refusal of parents/guardians to participate in this study (5 children). Males and females were of nearly equal distribution (47% vs. 53%, respectively). The mean age at examination was 54.4 months (SD 32.2). Most subjects were more than 1 year in age (87%). Median heart rate was 100 (78-140) beats per minute and median respiratory rate 28 per minute. No subject had fever at the time of examination (median temperature 36.8^{O}; range 36.4-37.5^{O} Celsius). Mean body weight was 14.3 kg (SD 5.6) and mean height was 96.4 cm (SD 19.1). Eighty-five percent of subjects had normal nutritional status and only 5 children were below the fifth percentile of the CDC-NCHS 2000 curve.

There were 21 (28%) children with clinical category A (mildly symptomatic), 28 (38%) in B (moderately symptomatic) and 25 (34%) in C (severely symptomatic). No subjects were in the N (not symptomatic) category (**Figure 1**). Based on CD4 percentage, subjects were classified into one of three categories: (1) without immunologic suppression (33 subjects or 45%), (2) moderate suppression (15 subjects or 20%), or (3) severe suppression (21 subjects or 28%). Almost half the subjects had CD4+ suppression at presentation, with median absolute CD4 cell count of 722.5 cells/mm^3 (range 2-3490 cells/mm^3). Ninety percent of children were on first-line ARV treatment, while none received protease inhibitors. No significant differences in age, weight, height, and body surface area were found among HIV clinical categories (P = 0.131, P = 0.346, P = 0.456, and P =



Note: A, B, C are HIV clinical categories; 1, 2, 3 are HIV immunological categories. Bar charts are percentage of total subjects

Figure 1. HIV Clinical and immunological categories of study subjects (n=74).

Table 1. Left ventricular dimension in HIV-infected children

Variables	Mean (SD)	95% CI
LV mass, g	47.5 (20.7)	42.7 to 52.3
LVEDD, mm	31.2 (5.9)	29.8 – 32.6
LVESD, mm	20.9 (4.7)	19.8 to 21.9
LVPWD, mm	5.9 (1.6)	5.5 – 6.2
LVPWS, mm	8.7 (1.7)	8.3 – 9.1
IVSD, mm	6.6 (1.4)	6.3 – 6.9
IVSS, mm	8.8 (1.5)	8.4 to 9.1

LV: left ventricle, LVEDD: left ventricular end diastolic dimension, LVESD: left ventricular end systolic dimension, LVPWD: left ventricular posterior wall diastolic thickness left ventricular posterior wall systolic thickness, IVSD: interventricular septum diastolic thickness, interventricular septum systolic thickness.

Table 2. Ventricular function in HIV-infected children

Variables	Mean (SD)	CI 95%
EF, %	62.8 (8.4)	60.8 to 64.7
FS, %	33.8 (5.6)	32.5 to 35.1
CO, L/minute	3.1(1.3)	2.7 to 3.3
CI, L/minute/m2	7.2 (2.2)	4.7 to 5.7
SV, ml	26.4 (10.6)	23.9 to 28.8
E, cm/second	100.4 (16.2)	96.7 to 104.2
A, cm/second	64.2 (13.9)	60.9 to 67.4
E/A	1.7 (0.5)	1.6 to 1.8
DT, seconds	0.135 (0.04)	0.126 to 0.143
TAPSE, mm	19.2 (3.9)	18.3 to 20.1

EF: ejection fraction, FS: fractional shortening, CO: cardiac output, CI: cardiac index, SV: stroke volume, E: MV E wave velocity, A: MV A wave velocity, DT: deceleration time, TAPSE: tricuspid annular plane systolic excursion

0.456, respectively) or in the HIV immunological suppression categories (P=0.325, P=0.726, P=0.35, and P=0.06, respectively).

No electrocardiogram abnormalities were found in our subjects. Echocardiography revealed dilatation of the left atrium and ventricle in 3 children, as well as mild pericardial effusion in 6 children. Neither moderate or large pericardial effusions, nor vegetations were found. LVFS below 25% was noted in 5 children (7%), while abnormal TAPSE was found in 16 children (22%). Three children who showed left-sided chamber dilatation took anti-cardiac failure drugs. Mean left ventricular systolic and diastolic function, right ventricular function, and mean left ventricular dimensions were within normal ranges, except for LVPWS and mitral valve peak E wave, which were higher than normal. **Tables 1 and 2** show the results of left ventricular dimensions and ventricular function for all subjects.

Assessment of the cardiac valves revealed no congenital anomalies. Trivial tricuspid regurgitation (TR) was found in 7 children, mild TR in 9, and moderate TR in 1 (peak gradient 34 mmHg). Mild mitral regurgitation was found in 5 subjects, trivial pulmonary regurgitation (PR) in 2, and mild PR in 2. We did not include 5 children in the analysis of ventricular dimension based on immunologic/CD4 status because their recent CD4 counts were not documented in the medical records. There were no significant differences in ventricular function and dimension between groups with and without CD4 suppression (**Tables 3 and 4**).

Subjects were divided into 3 groups based on HIV clinical category which consisted of categories A (21 children), B (28 children) and C (25 children). There were no statistically significant differences in LV function, RV function, LV dimension, and LV mass among the clinical categories (**Tables 5 and 6**).

Table 3. Ventricular dimension according to CD4 suppression

Variables	CD4 percentage		P	95% CI
	No suppression , n=33 Mean (SD)	With suppression, n=36 Mean (SD)		
LV mass, g	50.1(20.7)	46.1 (20.4)	0.169	-13.9 to 5.9
LVEDD, mm	31.9(6.4)	30.5(5.2)	0.323	-4.2 to 1.4
LVESD, mm	21.5(4.6)	20.3(4.1)	0.268	-3.3 to 0.9
LVPWD, mm	5.9(1.7)	5.9(1.6)	0.828	-0.8 to 0.7
LVPWS, mm	8.7(1.7)	8.7(1.7)	0.887	-0.9 to 0.7
IVSD, mm	6.7(1.2)	6.7(1.5)	0.883	-0.7 to 0.6
IVSS, mm	8.9(1.3)	8.7(1.6)	0.093	-0.8 to 0.5

LV: left ventricle, LVEDD: left ventricular end diastolic dimension, LVESD: left ventricular end systolic dimension, LVPWD: left ventricular posterior wall diastolic thickness left ventricular posterior wall systolic thickness, IVSD: interventricular septum diastolic dimension, interventricular septum systolic dimension.

Table 4. Ventricular function based on CD4 suppression

Variables	CD4 percentage		P	95% CI
	No suppression, n=33 Mean (SB)	With suppression, n=36 Mean (SB)		
FS (%)	34.1(5.2)	33.6(5.8)	0.169	-3.2 to 2.2
EF (%)	62.6(8.8)	63.1(7.8)	0.793	-3.5 to 4.5
SV (ml)	28.8(10.8)	24.3(9.8)	0.076	-9.5 to 0.5
CO (L/minute)	2.9(0.9)	3.1(1.5)	0.213	-0.5 to 0.7
CI (L/minute/m2)	4.9(1.8)	5.2(1.9)	0.421	-0.7 to 1.1
E (cm/second)	103.8(18.2)	97.2(14.6)	0.102	-14.5 to 1.3
A (cm/second)	63.5(12.9)	64.9(15.3)	0.302	-5.4 to 8.3
E/A	1.7(0.4)	1.68(0.54)	0.098	-0.26 to 0.19
DT (second)	0.144(0.04)	0.126(0.03)	0.051	-0.035 to 0.0004
TAPSE (mm)	19.9(3.3)	19.0(4.3)	0.329	-2.8 to 0.9

EF: ejection fraction, FS: fractional shortening, CO: cardiac output (L/minute), CI: cardiac index (L/minute/m^2), SV: stroke volume, E: E wave peak velocity, A: A wave peak velocity, DT: deceleration time, TAPSE: tricuspid annular plane systolic excursion

Table 5. Ventricular dimensions according to HIV clinical category

Variables	Clinical category			P	95% CI
	A (n=21)	B (n=28)	C (n=25)		
LV mass (g)	45.2(20.0)	48.4(21.3)	48.7(21.2)	0.829	-151 to 8.9
LVEDD (mm)	30.4(6.1)	32.7(5.7)	30.2(6.1)	0.236	-5.8 to 1.1
LVESD (mm)	19.5 (4,6)	22.3(4.6)	20.4(4.5)	0.105	-3.5 to 1.8
LVPWD (mm)	5.7(1.5)	5.8(1.6)	5.9(1.7)	0.872	-1.1 to 0.8
LVPWS (mm)	8.5(1.9)	8.9(1.6)	8.6(1.8)	0.724	-1.4 to 0.6
IVSD (mm)	6.7(1.3)	6.2(1.4)	6.9(1.4)	0.183	-0.3 to 1.2
IVSS (mm)	8.9(1.6)	8.4(1.5)	8.9(1.3)	0.335	-0.3 to 1.4

LV: left ventricle, LVEDD: left ventricular end diastolic dimension, LVESD: left ventricular end systolic dimension, LVPWD: left ventricular posterior wall diastolic thickness, left ventricular posterior wall systolic thickness, IVSD: interventricular septum diastolic dimension, interventricular septum systolic dimension.

Table 6. Ventricular function according to HIV clinical categories A, B, and C

Variables	Clinical category			P	95% CI
	A (n=21)	B (n=28)	C (n=25)		
FS	34.5(5.4)	32.2(5.7)	35.0(5.6)	0.157	-0.9 to 5.4
EF	62.7(9.6)	61.0(8.2)	64.8(7.2)	0.258	-3.1 to 6.4
SV	25.0(11.9)	27.5(10.5)	26.3(9.7)	0.725	-8.6 to 3.7
CO	2.8(1.1)	3.2(1.6)	3.0(0.9)	0.458	-1.18 to 0.3
CI	4.78(1.4)	5.0(1.9)	5.8(2.3)	0.170	-1.3 to 0.9
E	102.7(18.05)	97.6(14.4)	101.6(16.7)	0.511	-4.35 to 14.4
A	64.5(11.4)	63.3(12.1)	64.9(17.9)	0.910	-6.9 to 9.4
E/A	1.7(0.37)	1.68(0.4)	1.73(0.55)	0.874	-0.27 to 0.25
DT	0.143(0.04)	0.134(0.03)	0.127(0.03)	0.401	-0.01 to 0.03
TAPSE	19.2(3.4)	19.9(4.4)	18.3(3.6)	0.287	-2.9 to 1.5

EF: ejection fraction, FS: fractional shortening, CO: cardiac output, CI: cardiac index, SV: stroke volume, E: E wave peak velocity, A: A wave peak velocity, DT: deceleration time, TAPSE: tricuspid annular plane systolic excursion.

Discussion

The prevalence of LV dysfunction in this study was 7%. Only 3 children in the clinical category C showed dilated LA and LV, and only one had reduced FS and EF. Five children had FS below 25%, but we found no subjects with FS below 19%. Overall LV systolic function, LV mass, and cardiac dimensions in the 74 HIV-infected children in our study were in the normal range, according to normal reference values, except for LVPWS. Previous studies reported LV dysfunction prevalence at 5.7% for LVFS <25%, 3% for LVFS <19%, accompanied by clinical heart failure (CHF).[2] Al-Attar *et al*. reported that of children who died with AIDS, 35% had cardiac dysfunction.[8] Ten percent of children who had CHF required cardiac medications and 20% developed depressed LV function or LV dilatation during a 2 year observation.

The prevalence of LV enlargement in a previous study, indicated by increased LVEDD and LVESD, was found in 8.3% and 17.7% of children, respectively.[2] A previous study reported that the mean values for various cardiac parameters were significantly different from normal at baseline, and most remained so throughout follow-up, with some progressing.[7] The lower rate of LV dysfunction or enlargement in our study may relate to different population characteristics. Most of our subjects did not show any signs and symptoms of heart failure (subclinical heart impairment), while subjects of previous studies were mostly symptomatic, although they did not meet the CDC criteria for AIDS. Differences in number of subjects, sampling methods, and study design may also have influenced the results. The severity of LV dysfunction may be an important indicator for future survival.

Subclinical cardiovascular abnormalities are common in HIV-infected children. Previous data suggests that most FS reduction occurs during the first year in patients with previously normal LV function.[2,8] The majority of our subjects were below 10 years of age, with only 3 children above age 10 years, which

could have contributed to our differing results. Cardiac disease in HIV-infected children as an underlying cause of death shows a significant age-related trend. Death from cardiac disease is not seen in infancy, is unusual in early childhood, and increases to 25% of deaths in children above 10 years in age.[6] Follow-up checks are mandatory to investigate future cardiac problems in our study patients as they get older.

The course of HIV-infection may contribute to heart disease, beginning with a catastrophic condition affecting all organs, including the heart. ARV administration may improve cardiac condition.[4] Diseases with increased mortality associated with increased age are likely to become more important clinically as more patients survive their early years with the concomitant decreased risk of death from infection and pulmonary disease. Knowledge of specific age-related causes of mortality can guide efforts in the care of HIV-infected children.[6]

LV diastolic function in our study was in the normal range. Nor did we find abnormal relaxation or restrictive features which differed among HIV clinical and immunological categories. Despite the mitral valve E wave peak velocity being higher than the normal reference value, LV diastolic function was still preserved in these children. There had been no previous studies on diastolic dysfunction in HIV-infected children. Multiple factors associated with LA and LV that influence transmitral inflow could result in different diastolic function patterns. Further prospective study is required to assess diastolic function of HIV-infected children.

In the majority of our study subjects, RV function according to TAPSE evaluation was normal. Sixteen children (22%) had RV impairment with TAPSE <16 mm, but clinically they did not show signs and symptoms of RV dysfunction. Interestingly, RV dysfunction was more frequent than LV dysfunction. Associated factors that influence RV function are chronic and recurrent lung infection, and in fact, most of our subjects were on anti-tuberculosis treatment. Further studies are needed to find out what factors could contribute to these results. The TAPSE method to assess RV function has several limitations, including its single dimensional approach, which assesses regional RV function rather than global function. TAPSE is also influenced by cor pulmonale, RV strain, and respiratory cycle. However, TAPSE has advantages since it is a simple, easy procedure that is highly reproducible. Also, it correlates with RV ejection fraction, in that a dimension of <15 mm correlates with an RV EF of 40%.[13]

The LVPWS was higher compared to reference normal values. This finding was similar to previous studies suggesting that early increase of LVPWS may result from a normal but inadequate response to persistent LV dilatation. The ratio of LV thickness to dimension reduction results in increased peak wall stress. Elevated peak wall stress normally induces LV hypertrophy until the LV thickness-to-dimension ratio is adequate to normalize peak stress.[14] Persistent elevation of peak wall stress indicates an inadequate hypertrophic response to LV dilatation. Catecholamine in HIV-infected children has been reported to be increased, inducing LV hypertrophy associated with hyperdynamic LV function. Chronic catecholamine elevation, in fact may result in LV dysfunction.[1]

We found no significant differences in ventricular function and dimensions among CD4 level and HIV clinical categories. A previous study with a small number of subjects reported hypertrophy and dilatation of LV, found mostly in the C clinical category.[9] In children with AIDS, low CD4 was taken as a predictor of cardiac complications or cardiac death.[8] A previous study revealed no significant correlation between cardiac and immune dysfunction, even if there was a significant correlation between FS and CD4 cell count at baseline. However, the rate of FS decline did not correlate with advancing HIV infection.[7]

Several studies have shown that using CD4 cell count as a marker for clinical outcome has limitations. The wide gap between time of CD4 examination and cardiac assessment is one such limitation in our study. There was also discrepancy between clinical category determined at time of diagnosis and clinical condition at time of enrollment into this study. Most subjects had been improving clinically after ARV treatment. Early cardiac intervention also reduces subsequent cardiac morbidity and related mortality in HIV-infected children. The levels of lymphocyte subpopulations or combinations of surrogate markers, including the determination of viral load, may be more useful for determining the progression of cardiac disease in HIV-infected children.

Inflammation of the heart is commonly reflected by pericardial effusion, valve regurgitation, or ventricular dysfunction. A previous study reported that pericardial effusion occurred in 25% of HIV-infected children,[2] higher than that of our study (6 children or 8%). A study in 75 adults showed that 41% of patients had asymptomatic pericardial effusion, 13% moderate or severe, and 5.5% had moderate or severe effusions resulting in right atrium diastolic compression, some requiring pericardiocentesis. Most patients with moderate or severe effusion are not clinically distressed, but are at risk of life-threatening tamponade. Echocardiography is justified for surveillance in HIV-infected patients, especially for those with heart failure, Kaposi's sarcoma, tuberculosis, or other pulmonary infections.[15]

We found 17 children with moderate tricuspid regurgitation without RV dilatation, 5 children with mitral regurgitation, and 4 children with pulmonary regurgitation (PR). In children with HIV infection, the tricuspid valve is more commonly involved compared to the mitral valve. However, tricuspid regurgitation is not always related to pathological conditions, though sometimes it can be associated with pulmonary hypertension.[16] Yoshida *et al*. reported that in 40 normal children aged less than 10 years old, 45% had mitral regurgitation, 78% tricuspid regurgitation, 88% pulmonary regurgitation and none with aortic regurgitation. We also found no congenital heart disease in our subjects. A previous study reported no significant difference in the prevalence of congenital heart disease between HIV-infected children and those without infection, but born of HIV-infected mothers.[17]

Although the echocardiograms of our subjects were mostly normal, another study reported that echocardiography measures of LV structure and function are independent and potentially useful long-term and short-term predictors of overall mortality in HIV-infected children. Diagnosis of congestive heart failure may be difficult in children with HIV infection because other diseases also can cause tachycardia, tachypnea, and hepatomegaly, thus increasing reliance on clinical judgment of the caregiver.[2] Echocardiography measurement of LV structure and performance provides noninvasive, independent markers of disease and death in HIV-infected children that may be clinically useful. Serial echocardiograms of this population may identify children at risk who may benefit from more careful examination and interventions to alter the course of the disease.[18]

Fractional shortening may also be used as an important predictor for early death in HIV-infected children, as it represents the end result of multiple processes, including preload, after load, heart rate, and contractility.[3] Further study is needed to provide normal standard echocardiography references for Indonesian children. More studies are also required to determine whether early change in cardiac dimension in the baseline echocardiography of HIV-infected children is associated with increased cardiac morbidity and mortality and whether treatment of this kind of abnormality is beneficial.

In conclusion, the prevalence of LV dysfunction in children with HIV infection was 7% and RV dysfunction was 22%. There were no differences in LV dimension or ventricular function among HIV-infected children among the different HIV clinical or immunological categories. Further prospective studies are needed to assess cardiac problems and to develop a national, baseline cardiovascular database of children with HIV infection.

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