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HIV Cardiomyopathy in Manipur

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Abstract: *To assess the prevalence of cardiomyopathy in patients with HIV and its correlation with CD4 count and duration of disease.*
Materials And Methods: *A total of 50 HIV were selected randomly. Anthropometric parameters were recorded, age, CD4, BMI, duration of HIV and the echocardiographic examinations were performed in all patients according to standard techniques. Ejection fraction (EF) was calculated by the formula LVEF% = (LVID)2 – (LVIDS)2.*
Left ventricular EF was consider normal when EF was 55 to 75%. Diastolic dysfunction was calculated by measuring E and A transmitral inflow velocity. Left ventricular mass in grams is calculated by the formula: LVM(gm) =1.04x 0.8[(LVID + PWT + IVST)3 – LVID3]+ 0.6.
Results And Conclusion: *HIV Cardiomyopathy was found in 15 patients(30%) of the total study, 9 males(60%) and 6 females(40%).*
Key Words: *Cardiomyopathy, echocardiography , HIV.*

I. Introduction

Left ventricular dysfunction and dilated cardiomyopathy are common consequences of HIV infection in both adults and children. Cardiomyopathy carries a worse prognosis specially when it is associated with HIV infection. Signs and symptoms of heart failure in these patients may be atypical or masked by concurrent illness and malnutrition. ECG may reveal non specific conduction defects or repolarisation changes. Upto 5% of asymptomatic HIV infected patients have baseline ECG abnormalities like supraventricular and ventricular ectopics. Echocardiography is the only specific validated non invasive test for detecting left ventricular systolic dysfunction and diastolic dysfunction in this population. It often reveals increased left ventricular mass with low-normal or increased wall thickness and dilated left ventricle. Dilated cardiomyopathy is strongly associated with a low CD4 cell count. Clinical and echocardiographic findings suggested that diastolic dysfunction is relatively common in long terms survivors and usually precedes systolic dysfunction and has been reported in 8% of symptomatic HIV infected adults after 5 years of follow up.

Clinically apparent HIV cardiomyopathy may take years to develop, but echocardiography can detect significant abnormalities well before the onset of symptomatic heart failure. Early abnormalities are defined by the preserved left ventricular ejection fraction (EF) with reduced early diastolic filling. The diastolic precedes systolic dysfunction in HIV cardiomyopathy even before the presence of pathological findings on clinical examination. When ventricular relaxation is impaired, early diastolic filling decreases progressively and a vigorous compensatory atrial contraction ("a trial kick") occurs. With further disease progression, left ventricular compliance becomes reduced and filling pressures begin to increase. Systolic dysfunction occurs late when patients have already developed significant diastolic dysfunction. The prevalence of HIV cardiomyopathy varies between 20 to 40% according to different workers. Several studies have shown a correlation between CD4 and left ventricular diastolic dysfunction, whereas other studies have not found such correlation. Ethnicity is believed to play role in the development of heart failure, especially in association with HIV. Left ventricular dysfunction and cardiomyopathy is due to wasting and defiency of trace element.

II. Materials And Methods

A total of 50 HIV patients were selected from JNIMS, Imphal, Manipur. Patients who had acute or chronic complications of HIV illness, chronic alcoholism, carcinoma and any infection were excluded from the study.

A detailed history of the clinical information including the patient's age, gender, weight, height, and religion was noted. Body mass index(BMI) was calculated as weight in kilograms divided by height in meters square.

Echocardiography. The echocardiographic examinations were performed in all patients according to standard techniques while the patients were lying flat or in the decubitis position by Siemens Simatic Versa plus Colour Doppler ultrasound machine. Left ventricular measurements were made with the M mode beam positioned just beyond the mitral leaflets tips(mitral chordal level) perpendicular to the long axis of the ventricle. Standard

echocardiographic measurements of diastolic and systolic left ventricular dimension left ventricle internal dimension(LVID) , posterior wall thickness(PWT), and interventricular septum(IVS) were measured from the leading edge to leading edge of each interface of intersect for optimal measurement accuracy.

EF was calculated by the formula LVEF % = $(LVID)^2$ - $(LVIDS)^2$. Left ventricular EF was consider normal when it was between 55 to 75%. Diastolic dysfunction was calculated by measuring E and A velocity across transmitral inflow velocity. Left ventricular mass in grams was calculated by the formula LVM(gm) = $1.04 \times 0.8[(LVID + IVST + PWT)^3 - LVID^3] + 0.6$, where LVID is left ventricular end diastolicinternal diameter; IVST, interventricular septal thickness; and PWT, posterior wall thickness.

The diagnosis of HIV cardiomyopathy is made according to the following echocardiographic criteria.

i. Diastolic dysfunction was defined as preserved left ventricle EF with (a) reduced early diastolic filling, (b) prolongation off isovolumetric relaxation, (c) increased atrial filling, the presence of which confirms diastolic dysfunction, (d) increased pre-ejection period(PEP) and shorter left ventricular time(LVET) resulting in increased PEP/LVET ratio – an evidence of reduced left ventricular distensibility, and (e) left ventricular hypertrophy(LVH) on echocardiogram defined by LV mass ≥ 125 g/m^2 for men and ≥ 110 g/m^2 for women.
ii. Systolic dysfunction was characterized by reduced EF < 55%.

Results were given as mean ±SD. Means are compared by unpaires Students t-test. Chi-square or Fischer's exact test were used as appropriate. The observations and data were analysed in the statistical package social sciences(SPSS). The level of significance was set as $P<0.05$.

III. Results

HIV cardiomyopathy was found in 15 patients(30%) of the total study of 50 HIV comprising 9 males and 6 females.The duration of HIV was not significantly different between the cardiomyopathy groups, as shown in Table 1. The average BMI of all the patients with HIV was 22 The HIV with cardiomyopathy was found slightly more obese than those without cardiomyopathy($P>0.05$).

DCM: HIV Cardiomyopathy, IVST: Inter ventricular septum thickness,
IVSes: Interventricular septum end-systolic dimension,
LVPW: Left ventricular posterior wall thickness,
LVPWes: Left Ventricular posterior wall end-systolic dimension,
LVID: Left ventricular internal dimension,
LVIDes : Left ventricular internal dimension end systolic,
LV Mass: Left ventricular mass

Table 2 shows the echocardiographical characteristics of the study population. There were no significant differences in left ventricular septum IVS), LVID between the HIV with and without cardiomyopathy. A comparision of mean values of EF showed a significant reduction among the HIV cardiomyopathies compared with those without cardiomyopathies ($P<0.001$),however, the fractional shortening was found slightly higher among the HIV with cardiomyopathies than those without cardiomyopathy ($P<0.713$).

Left ventricular mass of the HIV patients with cardiomyopathy(250.457 + 97 g) was much higher than those without cardiomyopathy (177 ± 69.68). Although the left ventricular mass was larger for both males and females in patient with HIV cardiomyopathy counterparts(284 ± 136.8g vs 76g; $P<0.133$).

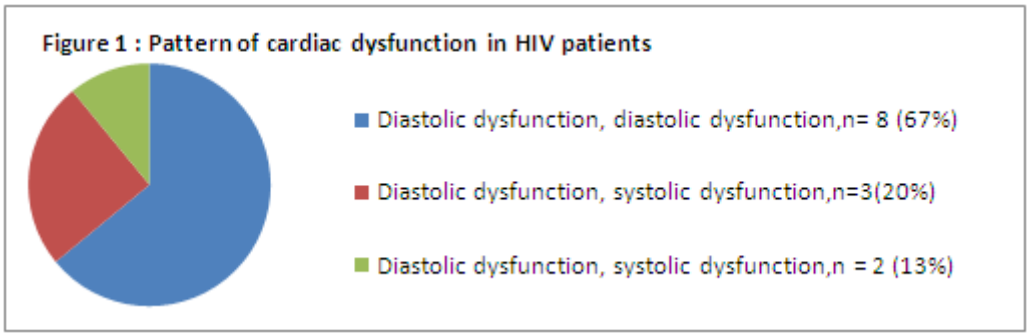
Among the 15(30%) patients with HIV cardiomyopathy in our study, 67% had left ventricular diastolic dysfunction, 20% had systolic dysfunction, and 13% had both systolic and left ventricular diastolic dysfunction. The incidence of diastolic dysfunction among HIV cardiomyopathy was found to be higher than systolic dysfunction($P<0.001$).

The incidence of diastolic dysfunction among HIV was found to be higher than systolic dysfunction($P<0.001$)

Table 1: Clinical characteristics

Clinical characteristics	DCM (n=15)	Non DCM (n=35)	P value
Age	40.32 ± 13.2	38.1 ± 11.79	o.4389
Sex(Male/Female)	13/7	36/14	
Duration in years	9.2 ± 3.3	8.2 ± 3.2	0.5324
BMI (kg/m^2)	22.6± 2.687	22.26 ± 2.35	0.5030
CD4	280 ± 21.2	420 ± 11.7	0.0007

Table 2: Echocardiographical characteristics			
	DCM (n=15)	Non-DCM (n=35)	P value
IVST(mm)	10.25 ± 3.99	9.7 ± 2.43	0.454
IVSes	12.7 ±3.770	12.35 ±2.94	0.604
LVPW(mm)	10.825 ±4.684	10.2 ±2.08	0.3654
LVPWes(mm)	14.92 ±6.086	13.98 ±1.93	0.2665
LVID(mm)	50.275 ±10.536	48.7 ±7.57	0.3865
LVIDes(mm)	43.25 ±12.996	43.91 ±14.55	0.9052
Ejection fraction(%)	50.1 ±19.577	61.21 ±7.60	0.0015
Fractional shortening(%)	33.075 ±19.888	32 ±8.91	0.7139
LV mass(mm)	250.475 ±97.467	177 ±69.78	0.0006



IV. Discussion:

In our study, 15 patients(30%) of 50 HIV had HIV cardiomyopathy of which 67% had left ventricular diastolic dysfunction, 20% left ventricular systolic dysfunction, and 13% had both. The incidence of left ventricular diastolic dysfunction among HIV cardiomyopathy was found to be significantly higher than systolic dysfunction (P<0.001) and patient with cardiomyopathy have lower CD4 count compared to patient without cardiomyopathy

(P< 0.008). EF among the HIV with cardiomyopathy is significantly lower compared with the HIV without cardiomyopathy(52.1 ± 19.58% vs 61.2 ± 7.6%, P< 0.001) in our study.

The left ventricular mass was more among HIV with cardiomyopathy compared with that of the HIV without cardiomyopathy, which is statistically significant(P=0.0099) and this is consistent with the finding of other workers. They opined that there is positive associations between heart weight and total fibrosis in patients with HIV alone and with both HIV and LVH. We can also observed that although the left ventricular mass was larger both in males and females with cardiomyopathy, it was much more so among the HIV females, which agrees with the findings of other workers.

V. Conclusion

Cardiomyopathy is a multifactorial disease initiated by wasting and deficiency of trace element leading to oxidative stress and extensive structure abnormalities. Clinically the process manifest as asymptomatic and later on progress to various cardiac dysfunction.

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