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ASSESSMENT OF LEFT VENTRICULAR SYSTOLIC/DIASTOLIC FUNCTION BY ECHOCARDIOGRAPHY IN SEROPOSITIVE HIV OTHERWISE ASYMPTOMATIC PATIENTS

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Abstract:
Acquired immunodeficiency syndrome is a serious problem worldwide. Recent advances in the knowledge about human immunodeficiency virus (HIV) replication and the treatment of HIV infection has improved survival in HIV patients. Because of the longer survival in HIV patients, the more manifestations of late-stage HIV infection will be seen, including HIV-related cardiac diseases. This may take the form of either a dilated cardiomyopathy or isolated left or right ventricular dysfunction, is associated with a poor prognosis, and results in symptomatic heart failure in up to 5% of HIV patients. The precise cause of HIV-associated cardiomyopathy remains unclear but is undoubtedly complex, and most probably multifactorial.
We Performed Case-Control study in 70 Seropositive asymptomatic HIV patients in SN medical College and found LV dysfunction (diastolic and systolic dysfunction) in comparison to age and sex-matched control group. Our study aims to find the cases of undiagnosed cardiac dysfunction in asymptomatic seropositive HIV cases in the early course of illness that are not on ART at yet so that they could be further monitored for cardiac dysfunction. We found a significant difference in LVEF in cases as compared to controls.

Keywords: Systolic and diastolic function, HIV patients, echocardiography.

Introduction:

Cardiac involvement in AIDS patient was first detected by Autran et al *(Autran B, Gorin L, Leibowitch M, et al. AIDS in a Hatitian woman with cardiac Kaposi's sarcoma and whipple disease. Lancet 1983;1: 767-768) in1983 who reported myocardial Kaposi sarcoma at autopsy. Because of longer survival in HIV patients, the more manifestation of late-stage HIV infection will be seen including HIV related cardiac diseases.

HIV-related heart muscle disease can present as a dilated cardiomyopathy, as isolated left ventricular dysfunction [1–3], or as non-specific right heart changes [3–6].

The prevalence of heart muscle disease in HIV-infected patients appears to be about 15% and longitudinal, and echocardiographic studies have confirmed that the development of either dilated cardiomyopathy or acute onset, symptomatic left ventricular dysfunction carries a poor prognosis in AIDS patients[7,8].

Material and methods

This study was carried out in S N Medical College and 70 Patients from ART center/OPD, S N Medical College were taken as cases in this study with following inclusion/exclusion criteria.

Inclusion Criteria:

- Seropositive for HIV
- CD4 count> 350
- Age< 45 yr
- Apparently asymptomatic patient

Exclusion Criteria:

Age>45 yr

- Smoker
- Heavy Alcoholic
- H/o HTN, DM
- H/o ART intake
- K/C/O Lung infection(TB, Pneumonia)
- K/C/O COPD and Pulm HTN

Mean age of cases- 34 +_ 5.3

Mean age of control 35+_ 7.1

Echo-2D & M-Mode & Doppler echocardiography was done in all cases and control group.

LV diastolic dysfunction was taken by mitral inflow velocity on Doppler where e (emptying of atria) & an (atrial contraction). Diastolic dysfunction is termed when e>a & Systolic Function was tested by M –mode echocardiography by calculating ejection fraction on the basis of the Teichholz method.

Contractility of LV wall was observed on 2D echocardiography on parasternal long axis view and short axis and apical view.

Same no of Age/sex/profile matched control group was taken as a control in this study.

These case & control both were examined clinically & Echocardiography was done where we took LV dysfunction – systolic & diastolic by mitral inflow velocity.

Observation:

Table 1: Distribution of patients selected as subjects for the study

Age	Cases N=70		Control N=70	
20-25 yr	8	5+3	10	7+3
25-30 yr	12	8+4	10	6+4
30-35 yr	23	13+10	20	10+10
35-40 yr	18	13+5	20	10+10
40-45 yr	9	4+5	10	5+5

Diastolic Dysfunction (e<a)

	Cases n=70	Controls n=70
	18	5

LVEF:

EF	Cases	Control
35-40%	6	0
40-45%	15	2

45-50%	13	3
50-55%	19	28
55-60% 60-65% >65 or hyperdynamic	12 2 3	20 12 5

< 50 case-28 control -5
<40 case-4 control- 0
Global hypokinesia was observed in 21 cases & in 2 control group.
Mean EF in both the group- Cases: 49.43% with SD +_ 7.45
Control: 55.71 with SD +_ 5.22.
T value is 6.246 which is highly significant at 0.01 level.

Discussion

The common cardiac manifestations in patients with acquired immunodeficiency virus are pericardial effusions, myocarditis, dilated cardiomyopathy, endocarditis, pulmonary hypertension, malignant neoplasms, and drug-related cardiotoxicity.

There are several hypotheses concerning the the etiology of HIV-related heart muscle disease, including:

(i) primary HIV myocarditis, direct infection with HIV; (ii) secondary HIV myocarditis, an indirect effect of HIV infection; (iii) opportunistic infection; (iv) nutritional deficiencies; and (v) toxic or drug-induced myocarditis.

The Dallas criteria for the histological diagnosis of myocarditis requires the presence of an inflammatory infiltrate of the myocardium with adjacent myocyte necrosis or degeneration that is not typical of ischemia [9].

Furthermore, significant numbers of infiltrating CD8 and CD45 lymphocytes have been found in association with increased major histocompatibility complex (MHC) class I antigen expression on apparently histologically normal endomyocardial biopsies from HIV-positive patients with cardiac failure [10].

Primar HIV Myocarditis: Although in vitro studies have shown that HIV is unable to enter skeletal myocytes [11], recent work on a newly developed human foetal cardiac myocyte cell line has

suggested that, despite the absence of CD4 cell receptors on myocytes, HIV-1 may be ingested through a specific Fc receptor [12]. Therefore, it remains possible that HIV could play a direct role in the pathogenesis of HIV-related heart muscle disease.

Secondary HIV myocarditis

HIV could damage myocytes through 'innocent bystander destruction' [13]. This hypothesis suggests that specific cells are damaged by proteolytic enzymes released through HIV replication in the interstitium, and may be particularly relevant to the myocardium as increased numbers of infected interstitial cells have been found in HIV-infected subjects with confirmed myocarditis [14].

It has also been postulated that HIV infection may lead to overproduction of aberrant interferon that, through the production of certain cytokines such as tumor necrosis factor (TNF) or interleukins (IL), stimulates destructive autoimmunity [15]. Indeed, HIV glycoprotein 120 has been shown to enhance IL-1_-induced nitric oxide production in neonatal rat cardiac myocytes, showing that further studies are needed to confirm this possible mechanism of cardiac damage in HIV infection [16].

IL-6, a multifunctional cytokine, was also found in excess in a small number of HIV-infected patients with proven and borderline myocarditis [17]

The levels also appeared to be inversely related to CD4 cell counts, and inducible nitric oxide synthase in particular correlated to a crude mortality rate and was most marked in those HIV patients with evidence of co-infection with CVB or CMV. Cytokine activation may thus play an important role in the pathogenesis of, and clinical course of, HIV-related heart muscle disease [18]

Bibliography:

1. Cohen IS, Anderson DW, Virmani R, et al. Congestive cardiomyopathy in association with the acquired immunodeficiency syndrome. N Engl J Med 1986, 315:628–630.
2. Corboy JR, Fink L, Miller WT. Congestive cardiomyopathy in association with AIDS. Radiology 1987, 165:139–141.
3. Stewart JM, Kaul A, Gromische DS, et al. Symptomatic cardiac dysfunction in children with human immunodeficiency virus infection. Am Heart J 1989, 117:140–144.
4. Lewis W. AIDS: cardiac findings from 115 autopsies. Prog Cardiovasc Dis 1989, 32:207–215.
5. Himelman B, Dohrmann M, Goodman P, et al. Severe pulmonary hypertension and cor pulmonale in the acquired immunodeficiency syndrome. Am J Cardiol 1989, 64:1396–1399.
6. Blanchard DG, Hagenhoff C, Chow LC, et al. Reversibility of cardiac abnormalities in human immunodeficiency virus (HIV)- infected individuals; a serial echocardiographic study. J Am Coll Cardiol 1991, 17:1270–1276.
7. Currie PF, Jacob AJ, Foreman AR, Elton RA, Brettle RP, Boon NA. Heart muscle disease related to HIV infection: prognostic implications. Br Med J 1994, 309:1605–1607.
8. De Castro S, D'Amati G, Gallo P, et al. Frequency of development of acute global left ventricular dysfunction in human immunodeficiency virus infection. J Am Coll Cardiol 1994, 24:1018–1024
9. Aretz TH, Billingham ME, Edwards WD, et al. Myocarditis: a histopathologic definition and classification. Am J Cardiovasc Pathol 1987, 1:3–14.
10. Herskowitz A, Wu TC, Willoughby S, et al. Myocarditis and cardiotropic viral infection associated with severe left ventricular dysfunction in late-stage infection with human immunodeficiency virus. J Am Coll Cardiol 1994, 24:1025–1032.
11. Clapham PR, Weber JN, Whitby D, et al. Soluble CD4 blocks the infectivity of diverse strains of HIV and SIV for T cells and monocytes but not for brain or muscle cells. Nature 1989, 337;368–370.
12. Herskowitz A, Willoughby S, Wu TC, et al. Immunopathogenesis of HIV-1 associated cardiomyopathy. Clin Immunol Immunopathol 1993, 68:234–241.
13. Ho DD, Pomerantz RJ, Kaplan JC. Pathogenesis of infection with human immunodeficiency virus. N Engl J Med 1987, 317: 278–286.
14. Rodriguez ER, Nasim S, Hsia J, et al. Cardiac monocytes and dendritic cells harbor human immunodeficiency virus in infected patients with and without cardiac dysfunction: detection by multiplex, nested, polymerase chain reaction in individually microdissected cells from right ventricular endomyocardial biopsy tissue. Am J Cardiol 1991, 68:1511–1520.
15. Skurkovich S, Skurkovich B, Bellanti JA. A disturbance of interferon synthesis

with the hyperproduction of unusual kinds of interferon can trigger autoimmune disease and play a pathogenetic role in AIDS: the removal of these interferons can be therapeutic. Med Hypotheses 1994, 42:27–35.

16. Kan H, Xie Z, Finkel MS. HIV gp120 enhances NO production by cardiac myocytes through p38 MAP kinase-mediated NFkappa _ activation. Am J Physiol Heart Circ Physiol 2000, 279:H3138–H3143.
17. Herskowitz A, Willoughby S, Wu TC, et al. Immunopathogenesis of HIV-1 associated cardiomyopathy. Clin Immunol Immunopathol 1993, 68:234–241.
18. Barbarao G, Di-Lorenzo G, Soldini M, et al. Clinical course of cardiomyopathy in HIV-infected patients with or without encephalopathy related to the myocardial expression of tumour necrosis factor-alpha and nitric oxide synthase. AIDS 2000, 14:827–838.