

CARDIOMYOPATHY INCIDENCE AND HIV DETECTION IN MYOCARDIAL CELLS

A prospective, long-term clinical study echocardiographically evaluated the incidence of dilated cardiomyopathy in HIV-positive patients ($n = 952$), correlating the clinical features with immunologic and virologic data. Clinical examinations were performed and CD4 cell counts were obtained every 3 months; electrocardiographic and echocardiographic examinations were performed every 6 months. Mean duration of follow-up was 60 ± 5.3 months. At the study's conclusion, echocardiographic diagnosis of dilated cardiomyopathy was made in 76 patients. Immunodeficiency influenced the incidence of dilated cardiomyopathy. Patients with a CD4 cell count less than 400 cells/mm^3 exhibited a higher incidence of dilated cardiomyopathy; in patients who received zidovudine, the incidence of cardiomyopathy was greater in patients with a CD4 cell count less than 300 cells/mm^3 . A histological diagnosis of myocarditis was made in 63 patients with dilated cardiomyopathy. HIV nucleic acid sequences were detected in 76% of the patients with dilated cardiomyopathy and in 57% of the patients with a histologic diagnosis of myocarditis. The study concluded that HIV has a direct action in inducing myocarditis and that there is a pathogenetic relation between myocarditis and dilated cardiomyopathy. Further studies are necessary to understand the pathogenesis of cardiomyopathy and myocarditis in both HIV-infected and non-infected patients.

Barbaro G, Di Lorenzo G, Grisorio B, Barbarini G: Incidence of dilated cardiomyopathy and detection of HIV in myocardial cells of HIV-positive patients. N Engl J Med 1998;339:1093-1099.

HIV-1 INFECTION, IMMUNOSUPPRESSION, AND INFECTIVE ENDOCARDITIS IN INTRAVENOUS DRUG USERS

A prospective cohort study evaluated intravenous drug users diagnosed with infective endocarditis (IE) to determine whether HIV infection and the degree of HIV-induced immunosuppression changed the clinical characteristics and prognosis of IE. Patients ($n = 283$) with IE and known HIV serostatus were included in the study; 216 (76.3%) were infected with HIV, and 67 (23.7%) were non-infected patients. Several characteristics were independently associated with HIV infection. Chest pain, pulmonary infiltrates, and skin lesions suggestive of systemic embolism were more common in HIV-infected patients. Leukocyte, neutrophil, lymphocyte, and platelet counts were significantly lower in the HIV-infected patients. Finally, right-sided IE as well as *Staphylococcus aureus* infection were more prevalent in the HIV-infected patients. There was no significant difference in mortality

caused by IE between the HIV-infected and non-infected patients. The study concluded that HIV status influences presentation and outcome of IE in intravenous drug users. HIV-seropositive intravenous drug users with IE had been addicted longer, had a higher incidence of right-sided IE, and had lower leukocyte counts compared to intravenous drug users who were HIV-seronegative.

Ribera E, Miro JM, Cortes E, et al: Influence of human immunodeficiency virus 1 infection and degree of immunosuppression in the clinical characteristics and outcome of infective endocarditis in intravenous drug users. Arch Intern Med 1998;158:2043-2050.

CERVICAL AND VAGINAL HIV-1 CELL SHEDDING THROUGHOUT THE MENSTRUAL CYCLE

A prospective study evaluated the cervical and vaginal secretions of 17 HIV-1-seropositive women, with specific attention to the cervical and vaginal shedding of HIV-1-infected cells in relation to plasma HIV-1 RNA levels, phase of the menstrual cycle, serum estradiol and progesterone levels, and other factors. Patients were examined daily over the course of one menstrual cycle. Cervical and vaginal swab samples and urine samples were collected daily; blood was drawn three times per week for determination of serum estradiol and progesterone levels. HIV-1 DNA was detected in 207 (46%) of 450 endocervical swab samples and 74 (16%) of 449 vaginal swab samples. There was a significant association between vaginal and cervical HIV-1 DNA shedding: vaginal HIV-1 DNA was detected on 61 of 206 days on which endocervical HIV-1 DNA was detected and on 13 of 243 days on which endocervical HIV-1 DNA was absent. However, there was no pattern of shedding of HIV-1-infected cells by day or phase of the menstrual cycle. Neither serum estradiol nor progesterone was significantly associated with cervical or vaginal HIV-1 DNA detection. The study concluded that there is substantial variability in cervical and vaginal shedding of HIV-1-infected cells among women and that the pattern of shedding is not significantly influenced by menstrual cycle and hormonal changes. Additional studies are necessary in order to understand the relationship between the HIV-1 subtype and genital tract shedding.

Mostad SB, Jackson S, Overbaugh J, et al: Cervical and vaginal shedding of human immunodeficiency virus type 1-infected cells throughout the menstrual cycle. J Infect Dis 1998;178:983-992.

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