

PNEUMONIA AND HIV DISEASE PROGRESSION

A cohort study examined HIV-positive patients with bacterial pneumonia or *Pneumocystis carinii* pneumonia (PCP) and compared them with control patients in order to determine whether specific opportunistic infections act as cofactors in HIV disease progression and whether the two types of HIV-related pneumonia have different impacts on incidence of subsequent HIV-associated disease and survival. Survival times of the two groups of patients with pneumonia were compared with survival times of the two control groups. The incidence of opportunistic infection and HIV-associated neoplasm in both the pneumonia and control patient populations was also compared. One-hundred fifty patients with at least one diagnosed episode of bacterial pneumonia were identified; 299 control patients were matched to pneumonia patients with corresponding CD4 lymphocyte counts (two controls per pneumonia patient for all but one). One-hundred fifty-two patients were diagnosed with PCP; 280 control patients were matched with the PCP patients according to CD4 lymphocyte counts (at least one control per PCP patient). Rates of HIV-associated opportunistic infection and neoplasm were nearly twofold higher among the patients with pneumonia compared with the control patients. Patients with bacterial pneumonia or PCP had significantly shorter survival times compared with the control patients. Median duration of survival was 24 months for patients with bacterial pneumonia compared with 37 months for control patients and 23 months for patients with PCP compared with 30 months for control patients. The study concluded that both bacterial pneumonia and PCP are associated with increased morbidity and shortened survival in HIV-positive patients compared with the rates for HIV-positive patients without either type of pneumonia.

Osmond DH, Chin DP, Glassroth J, et al: Impact of bacterial pneumonia and Pneumocystis carinii pneumonia on human immunodeficiency virus disease progression. Clin Infect Dis 1999;29:536-543.

CALL FOR LOWER CLINICAL THRESHOLD FOR HIV TESTING

An article addressed issues related to HIV testing, including the timing of clinical presentation of patients infected with HIV, the status of HIV testing in the United States, the seroprevalence of HIV infection in specific populations, the physician's role in HIV testing, screening recommendations, and clinical triggers that can lead to HIV screening. Policy issues related to HIV testing were also discussed. The article stated that initiation of medical care early in the course of HIV infection is dependent upon early testing; however, HIV testing is frequently delayed. Aggressive HIV testing has been advocated, but a passive approach

prevails in many clinical settings because physicians generally view HIV testing as a diagnostic tool rather than an approach to screening. Any physician encounter may provide an opportunity to test a patient for HIV infection. Primary care clinicians routinely screen for diseases with lower prevalence than HIV infection, including diseases for which the benefits of therapy have been less clearly demonstrated (eg, Papanicolaou smear for cervical cancer). With the availability of highly active antiretroviral therapy, not testing for HIV means not providing effective therapy to patients who may benefit substantially. The article concluded that the appropriate clinical threshold for HIV testing must be lowered. In order to have an impact on the HIV epidemic, physicians must be more vigilant and a screening paradigm for HIV testing must be developed.

Freedberg KA, Samet JH: Think HIV. Why physicians should lower their threshold for HIV testing. Arch Intern Med 1999;159:1994-1999.

VIRAL AND IMMUNOLOGIC EXAMINATION OF HIV-1-INFECTED SERONEGATIVE PATIENTS

Six patients with AIDS-defining symptoms and virally detectable HIV-1 infection who remained persistently seronegative for HIV-1 antibody (HIPS) were examined to determine a possible reason for the lack of an HIV-specific antibody response. Cells and plasma from the six HIPS patients were gathered and analyzed. All six patients had low CD4 cell counts and various opportunistic infections; patients were all p24-antigen positive and had plasma virus loads ranging from 17,000 to more than 4 million copies/mL. Western blot analysis revealed that three HIPS patients had weak p24 antibody reactivity before dissociation. None of the patients revealed any HIV-1-specific IgA responses in plasma. Virologic analysis suggested that the patients had no specific changes in the examined structural or regulatory gene regions that could explain the lack of antibody responses. The study concluded that the lack of detectable antibody response may be a result of immune dysfunction. Whether the immune dysfunction is caused by an imbalance in the Th1- and Th2-type responses, a selective loss of memory cells, a defect in the antigen-presenting cell, an induction of suppressor cells and factors, or an elaboration of cytokines that exhibit immunoregulatory properties is unknown.

Ellenberger DL, Sullivan PS, Dorn J, et al: Viral and immunologic examination of human immunodeficiency virus type 1-infected, persistently seronegative persons. J Infect Dis 1999;180:1033-1042.

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