EARLY THERAPY WITH HYDROXYUREA, DIDANOSINE, AND A PROTEASE INHIBITOR

Patients (n = 10) with a 1-year or shorter history of HIV infection were placed on a drug regimen of hydroxyurea, didanosine, and indinavir prior to complete Western blot (WB) seroconversion; the effect of early initiation of treatment was analyzed. After 46 ± 21 weeks of therapy, changes in CD4 and CD8 cell count and the CD4/CD8 ratio were +154, -544, and +0.46, respectively. HIV was below the level of detection in the plasma. Complete WB seroconversion eventually developed in most patients; however, overall progression of WB seroconversion was very slow. Blood from six of the 10 patients was examined to determine if the early treatment regimen confined the size of the HIV reservoir. An ultra-sensitive assay detected low levels of the virus in five patients; no HIV was isolated in the sixth patient, even when 37 × 10^6 cells were analyzed. The study concluded that early treatment may have a major effect on the outcome of HIV infection. Evidence supports that the earliest treatment may lead to the fastest recovery of the naïve cell repertoire and also may limit the establishment and expansion of latent HIV reservoirs.


PREVALENCE OF LOWER GENITAL TRACT INFECTIONS

A longitudinal cohort study assessed the prevalence of lower genital tract infections in HIV-seropositive and high-risk HIV-seronegative women to determine the relationship between lower genital tract infections and HIV serostatus, immunologic parameters and limited viral reservoir.


TRIMETHOPRIM-SULFAMETHOXAZOLE RESISTANCE

A serial cross-sectional study examined the development of resistance to trimethoprim-sulfamethoxazole (TMP-SMX) in relation to HIV infection in inpatients and outpatients at San Francisco General Hospital (San Francisco, CA) between 1979 and 1995. Patients whose microbiologic specimens were culture-positive for Staphylococcus aureus or one of seven common Enterobacteriaceae genera were included in the study. TMP-SMX resistance was stable (2% to 5.5%) between 1979 and 1986, increased slightly to 7.2% in 1988, and then markedly increased, reaching 20.4% by 1995. The rapid increase in TMP-SMX resistance temporally coincided with an increase in prophylactic TMP-SMX use in San Francisco as measured in the Pulmonary Complications of HIV Infection Study. Between 1988 and 1991, isolates obtained from HIV and non-HIV units had an equivalent and relatively stable prevalence of TMP-SMX resistance. However, prevalence of TMP-SMX resistance in isolates collected from HIV units increased from 6.3% in 1988 to 53% in 1995. Isolates obtained from the non-HIV units demonstrated an increase from 7.3% in 1988 to 17.7% in 1995. In terms of individual genera, isolates from HIV-infected patients demonstrated a significantly higher prevalence of TMP-SMX resistance (eg, TMP-SMX resistance in S. aureus increased from 0% to 48% from 1988 to 1995). Although not clearly supported by the data, the study concluded that long-term oral TMP-SMX use in HIV-infected patients is a potential cause of increased TMP-SMX resistance.