ı cellular responses to Haemophilus ducreyi in the presence or absence of HIV infection.

King, R; Choudhri, SH; Nasio, J; Gough, J; Nagelkerke, NJ; Plummer, FA; Ndinya-Achola, JO; Ronald, AR

Abstract:

We aimed to determine if the clinical and histological features of chancroid are altered by HIV infection. Male patients presenting to the Nairobi special treatment clinic with a clinical diagnosis of chancroid were eligible for the study. A detailed history, physical examination, swabs for Haemophilus ducreyi culture and blood for HIV serology, syphilis serology and CD4 counts were obtained from all patients. Punch biopsies from an ulcer were obtained from 10 patients and either fixed in 10% formalin or snap frozen in Optimum Cutting Temperature (OCT) medium compound at -70 degrees C. Patients were treated with erythromycin and followed for 3 weeks. Chi-square and Student's t-test were used to determine if the clinical and laboratory features of chancroid differed between HIV-seropositive and seronegative individuals. Cox regression survival analysis was used to determine if HIV infection altered cure rates of chancroid at 21 days. Immunohistochemical staining was performed using lymphocytic and macrophage markers and tissue sections were analysed by 2 pathologists in a blinded manner. Between February and November 1994, 109 HIV-seropositive and 211 HIV-seronegative individuals were enrolled in the study. HIV patients had ulcers of longer duration than HIV-seronegative patients (P=0.03). Although cure rates were similar at 3 weeks, HIV patients had lower cure rates at 1 week (23% v 54%, P=0.002). A dense interstitial and perivascular inflammatory infiltrate extending from the reticular to deep dermis was present in all biopsies. This consisted of equal amounts of CD4 and CD8 T-lymphocytes as well as macrophages. The histological and immunohistochemical picture was identical for HIV-positive and negative patients. HIV infection slows the healing rates of chancroid ulcers despite appropriate antibiotic therapy. This clinical difference cannot be attributed to an altered histopathological response to HIV infection. Additional studies are needed to elucidate the mechanisms responsible for this finding. PIP: Chancroid is caused by infection with Hemophilus ducreyi, and is associated with an increased risk for the sexual transmission of HIV-1. The authors assessed whether the clinical and histological features of chancroid are changed by HIV infection, using 320 male patients who presented during February-November 1994 to the City of Nairobi Special Treatment Clinic with a clinical diagnosis of chancroid. 109 subjects were HIV seropositive and 211 were HIV seronegative. A detailed history, physical examination, swabs for Hemophilus ducreyi culture and blood for HIV serology, syphilis serology, and CD4 counts were obtained from all patients. Punch biopsies from an ulcer were obtained from 10 patients and either fixed in 10% formalin or snap frozen in Optimum Cutting Temperature (OCT) medium compound at -70 degrees Celsius. Patients were treated with erythromycin and followed for 3 weeks. HIV patients had ulcers of longer duration than did HIV-seronegative patients. Although cure rates were similar at 3 weeks, HIV patients had lower cure rates at 1 week (23% vs. 54%). A dense interstitial and perivascular

the reticular to deep dermis was present in all biopsies.
s of CD4 and CD8 T-lymphocytes as well as
macrophages. The histological and immunohistochemical picture was identical for HIV-positive and HIV-negative patients. Study findings therefore indicate that HIV infection slows the healing rates of chancroid ulcers despite appropriate antibiotic therapy. The clinical difference cannot be attributed to an altered histopathological response to HIV infection