was 52,900 copies/mL, and his plasma level of HCV RNA (Amplicor HCV Monitor; Roche Diagnostics, Branchburg, NJ) was 132,052 copies/mL.

The strains of HIV and HCV that infected the 2 patients were compared. Viruses were isolated from the plasma of patient A at 4 months after the incident and from patient B at 6 months after the incident. Both HCV strains were determined to be genotype 2a. Nucleotide sequencing of the amplification product of the V3 region of HIV and the NS5b region of HCV revealed, respectively, 98.7% and 100% identity for the strains infecting the 2 patients. Fifteen months after the fight, the serological results of HTLV-1 testing were still negative.

Simultaneous transmission of either HIV and HCV or HIV and HBV from a single source has been previously described [2, 3]; however, to our knowledge, this is the first proven case of HIV-HCV coinfection that occurred as the result of a blow with the fist. Although HIV is probably infrequently transmitted via this route, this case raises the question of whether prophylaxis should be used after potential exposure to HIV during a bloody fight with an HIV-infected (or possibly HIV-infected) individual, as is recommended after other types of potential exposure to HIV, especially among individuals with frequent occupational exposure to HIV (e.g., police and fire department employees, etc.) [4]. Similarly, the risk of transmission of HCV infection during violent incidents should be taken into account.

Sylvie Abel,1 Raymond Césaire,2 Danielle Caules-Quist,1 Odile Béra,1 Guy Sobesky,1 and André Cabè1

1Center for Information and Care on Human Immunodeficiency, and 2Laboratory of Virology and Cellular Immunology, University Hospital of Fort-de-France, Martinique, French West Indies

Active Tuberculosis in Individuals Infected with Human Immunodeficiency Virus after Isoniazid Prophylaxis

A review was conducted in Haiti to determine the timing and outcome of active tuberculosis (TB) in human immunodeficiency virus (HIV)–positive patients who had previously received isoniazid (INH) prophylaxis. Of 1005 HIV-seropositive patients who completed INH prophylaxis, 14 (1.4%) subsequently had active TB diagnosed. The median interval between discontinuation of INH prophylaxis and TB diagnosis was 8 months for 6 patients receiving 6 months of INH, 22 months for 5 patients receiving 12–24 months of INH, and 40 months for 3 patients receiving 24–36 months of INH (P = .026). There is a postprophylaxis effect on INH that is dependent upon the duration of therapy.

Primary isoniazid (INH) prophylaxis is recommended for HIV-infected patients who have a positive tuberculin skin test but do not have active tuberculosis (TB) [1]. Studies have shown that INH prophylaxis for HIV-positive patients who have a positive tuberculin test decreases the risk of active TB and prolongs survival [2–4]. INH regimens of 6 and 12 months’ duration have both been shown to be effective, but to our knowledge, the efficacy of the 2 regimens has never been compared, and the optimal duration of prophylaxis remains unknown.

Although active TB is rare after INH prophylaxis, cases do occur. In Haiti, the incidence of active TB after 1 year of INH prophylaxis is 1.7 cases per 100 person-years [2]. We conducted a study to determine the timing and outcome of active TB in HIV-infected patients who have received INH prophylaxis.

The study was a retrospective review of cases seen at the treatment center run by the Haitian Study Group on Kaposi’s Sarcoma and Opportunistic Infections (GHESKIO) in Port au Prince, Haiti. GHESKIO is a national center for voluntary counseling, testing, and care of patients infected with HIV, and provides free service to a poor urban population. Antiretroviral therapy is unavailable to the economically disadvantaged population served by the GHESKIO center. HIV-infected patients are screened for active TB by use of a PPD test, chest radiograph, and a sputum smear and culture. Patients who are HIV seropositive and PPD reactive, but who do not have active TB, receive INH prophylaxis. The duration of INH prophylaxis varies, at the discretion of the physician and the patient, from 6 months to 3 years.

HIV-seropositive adults (>18 years of age) who had active

References
TB diagnosed after they had discontinued INH prophylaxis were eligible for this study. Case patients were detected by cross-referencing a register of patients with TB diagnosed from 1992 through 1998 with a pharmacy record of patients who received INH prophylaxis. Patients who developed active TB before they discontinued INH prophylaxis were excluded from this analysis.

We defined a case of active TB on the basis of the definition of the American Thoracic Society [5]. We required that at least 2 of the following 3 criteria be met: (1) clinical symptoms of TB (cough, fever, night sweats, etc.); (2) acid-fast bacteria (AFB) detectable in sputum samples, or Mycobacterium tuberculosis (MTB) cultured from sputum samples; and (3) a chest radiograph independently interpreted as highly suggestive of TB. For patients for whom there was no microbiological confirmation, we also required a clinical response to antituberculous medications. Patients were staged according to the 1993 HIV classification system of the Centers for Disease Control and Prevention [6].

Examination for AFB was performed by use of Ziehl-Neelsen staining. Culture for mycobacteria was performed in Lowenstein-Jensen medium after NaOH digestion, decontamination, and concentration of sputum. Antibiotic sensitivity testing of M. tuberculosis was performed at the New York Hospital–Cornell Medical Center. Differences in proportions were determined by use of Fisher’s exact test. Analysis of variance of >2 groups was done by use of the Kruskal-Wallis test for nonparametric data.

From 1992 through 1998, 1005 HIV-seropositive individuals completed INH prophylaxis at the GHESKIO center, and in 14 of these (1.4%), TB was subsequently diagnosed. Of these 14 patients, 6 received INH prophylaxis for 6 months, 5 patients received INH for 12–24 months, and 3 patients received INH for 24–36 months before the diagnosis of active TB. There were no significant differences in baseline characteristics between these subgroups with respect to age, sex, or stage of HIV at time of initiation of prophylaxis. Eight (57%) of the 14 patients were women. The median age at the time of TB diagnosis was 39 years. Six (43%) of the 14 patients had an AIDS-defining illness before the diagnosis of active TB. The median CD4 count for the 14 patients at the time of TB diagnosis was 240 cells/mm³. For 11 (79%) of the 14 patients, an AFB smear and/or a culture for MTB gave a positive result; for 3 (21%), the smear and culture gave negative results, and TB was diagnosed on the basis of clinical criteria.

For all 14 patients, the median interval from the discontinuation of INH prophylaxis to the diagnosis of active TB was 21 months. For the 6 patients who received INH prophylaxis for 6 months, the time intervals were 3, 4, 7, 9, 13, and 23 months (median, 8 months); for the 5 patients who received 12–24 months of prophylaxis, the intervals were 4, 20, 22, 33, and 43 months, (median, 22 months); and for the 3 patients who received 24–36 months of prophylaxis, the intervals were 36, 40 and 59 months, (median, 40 months; P = .026; figure 1.)

All patients started receiving an anti-TB drug regimen that included rifampin. Of the 14 patients, 7 (50%) were cured of TB, 1 (8%) responded to therapy initially but developed recurrent TB 18 months later, 3 (21%) failed therapy, and 3 (21%) died during therapy. We found no relationship between the treatment outcome and either the duration of INH prophylaxis or the treatment regimen used. Of the 3 patients who failed therapy, 2 had their MTB isolates tested for antibiotic sensitivity, and these isolates were resistant to INH and rifampin. Sensitivity testing was not performed for the other patients.

INH prophylaxis for TB appears to have an effect on later TB infection that is dependent upon the duration of the prophylaxis. Single-drug prophylaxis in immunocompromised individuals may decrease the number of mycobacteria but may not eradicate all of the microorganisms. One can postulate that a longer duration of INH prophylaxis may kill a larger fraction of the mycobacteria and thereby delay the subsequent development of active TB.

To our knowledge, there has never been a direct comparison of 6-month and 12-month INH prophylaxis regimens for HIV-positive individuals. However, a review of the literature for HIV-negative immunocompetent individuals suggests that a 12-month regimen may be superior to a 6-month regimen [7]. Although reports suggest that short-course multidrug regimens and a 12-month regimen of INH have similar efficacy [8], multidrug regimens are often more expensive than INH treatment and may be unavailable for people in developing countries, where INH is often the only prophylactic drug available. However, the optimal duration of INH prophylaxis remains unknown. We believe that the postprophylaxis effect documented in this report provides an argument for a longer duration (>12 months) of INH prophylaxis for HIV-positive patients.

The patients in this review responded poorly to TB treatment.

![Figure 1. Median interval between discontinuation of isoniazid (INH) prophylaxis and the diagnosis of tuberculosis (TB) in HIV-infected patients, according to the duration of INH prophylaxis.](https://example.com/figure1.png)
Nearly 50% of the patients either had therapy failure or died before completing therapy. We postulate that patients did not respond to therapy because they were severely immunocompromised at the time of TB diagnosis. Although INH prophylaxis prevents active TB in most HIV-positive patients, in a few individuals it may postpone the development of active TB until late in the course of HIV disease. Previous studies have shown that an advanced stage of HIV disease at the time of TB diagnosis is associated with poor treatment outcome and death [9–11]. Nearly one-half of the patients in this review had an AIDS-defining illness before the diagnosis of TB; their median CD4 count was 240 cells/mm³. This is in contrast to other patients followed in our clinic who had not received INH prophylaxis, in whom TB developed early in the course of HIV disease [12].

Poor treatment outcome may also have been due to drug resistance. Of note, 2 of the patients who failed therapy had multidrug-resistant strains of MTB. However, for this report, we have insufficient data on drug resistance to adequately address this issue.

Our study was a retrospective case review, and therefore the results must be interpreted cautiously. As the duration of INH prophylaxis was not chosen at random for the patients in this study, factors involved in those choices may have confounded our results. Furthermore, patients were not followed prospectively, and we cannot confirm that the duration or thoroughness of follow-up was the same for all patients. However, we believe that our data suggest that INH prophylaxis has an effect on later TB infection that depends on the duration of the prophylaxis, and we thereby suggest that there is an advantage to long-term INH prophylaxis.

Daniel W. Fitzgerald,¹,² Michelle Marie Morse,³
Jean William Pape,¹,² and Warren D. Johnson, Jr.²
¹Groupe Haïtien d’Étude du Sarcome de Kaposi et des Infections Opportunistes, Port au Prince, Haiti; ²Cornell University Medical College, New York, New York; and ³Johns Hopkins University School of Medicine, Baltimore, Maryland

Adipose Redistribution in Human Immunodeficiency Virus–Seropositive Patients: Association with CD4 Response

We have noted that human immunodeficiency virus (HIV)-seropositive patients who develop adipose redistribution (AR) while receiving combination antiretroviral therapy (ART) also tend to have a better response to ART, as indicated by the relative change in CD4 percent compared to those who don’t develop AR on therapy. Whether the possible benefits of the improved CD4 response outweigh the metabolic and cosmetic problems associated with lipodystrophy is not known.

Adipose redistribution (AR; also called “lipodystrophy”) has been widely reported among HIV-seropositive patients treated with combination antiretroviral therapy (ART) [1–6]. The negative aspects of AR, which include problems with cosmetic appearance and an association with dyslipidemia and insulin resistance, have been emphasized, as measured by CD4 count, compared with those who do not develop AR. We have noted that patients who develop AR also tend to have a better response to therapy, as measured by the CD4 count. We compared the CD4 response and the HIV viral load response to ART in patients who developed AR versus those who did not. Although AR has been reported in patients who were following

References