LETTER TO THE EDITOR

HIV Infection and Loss of Treponemal Test Reactivity

Sir,

Reactivity in the serological treponemal specific tests are considered to stay with the patient for the rest of his lifetime as a marker of a previous disease infection (1, 2). However, it was recently reported that in HIV-positive patients this rule of thumb does not always apply (2). To confirm this observation, and to supply some additional data on the humoral immune status during this negative seroconversion, we wish to report the following case.

In July 1986, a 34-year-old man was referred to the STD clinic because of a rash arousing suspicion of secondary syphilis. Reagin tests revealed high antibody titres (Wasserman complement fixation was positive at the maximum serum dilution tested, 480), and VDRL showed a very strong aggregation), and the specific treponemal tests (TPHA, FTA-abs and TPI) were clearly positive as well. He was also noted to be HIV-positive. Penicillin was administered intramuscularly (2.25 million IU daily for 17 days).

During the next two years the patient showed no signs and had no symptoms from his HIV infection, other than swollen lymph nodes. Initially the T-helper lymphocyte count was only slightly lowered (August 1986, 0.25 × 10⁹/l; July 1987, 0.30 × 10⁹/l). After about two years it fell dramatically (from May 1988 and onwards 0.01–0.05 × 10⁹/l). In April 1988, Pneumocystis carinii pneumonia (and AIDS) was diagnosed and the patient was treated with pentamidine inhalations. In November 1989 Kaposi's sarcoma was diagnosed and he was given vincristine and vinblastine, and later zidovudine (AZT).

The Wasserman and VDRL titres gradually fell to become negative one year after syphilis was diagnosed. No specific testing was performed at these later occasions. However, in April 1988 he was noted to be negative in TPHA. This non-reactivity was demonstrated also for TPI and has later been confirmed on numerous occasions. However, he remained HIV antibody-positive. Using frozen sera saved upon earlier visits, it was shown that the non-reactivity stage of the treponemal tests occurred between August 1986 and April 1988, i.e. before AIDS was diagnosed. Sera from 1986, 1988 and 1989 were tested for rubella-IgG, cytomegalovirus-IgG, varicella zoster-IgG and also for antibodies to streptolysin O (AST) and staphylococcal α-hemolysin (ASTA). In all of these tests a measurable antibody level was seen, with no significant change occurring between 1986 and 1989. The overall IgG level was slightly elevated (24, 18 and 19 g/l, respectively). For these reasons, the TPHA- and TPI-negative seroconversion observed in this patient does not seem to have been linked to a serious humoral immune derangement caused by HIV and/or the medications prescribed.

In HIV-positive patients, therefore treponemal tests need to be regarded as unsensitive markers of previous syphilis infection.

REFERENCES


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