

"Gay bashing" as possible risk for HIV infection

SIR,—Transmission of HIV is primarily through intimate sexual contact with an infected individual or contact with infected blood via shared needles among intravenous drug users, occupational exposure to contaminated "sharps", or transfusions with contaminated blood products, for example. We present a case of possible HIV infection via a previously undescribed route.

A 49-year-old heterosexual male was referred for evaluation of HIV seropositivity found on screening during a routine application for life insurance. A repeat ELISA and subsequent western blot were both reactive for HIV. The patient had been married for over 25 years and had three children. His wife's serum was non-reactive for HIV antibody. He denied ever having sex with a man, nor with another woman since marriage, and claimed he had been impotent for about 10 years. He denied ever having received blood products. He had used intravenous drugs once, 3 years previously, but distinctly remembered not sharing needles but using a needle from an unopened package. On repeated questioning he adamantly denied using drugs on any other occasion. This was all independently corroborated by his wife.

On a follow-up visit the patient, dissatisfied with the explanation that the alleged one-time exposure to intravenous drugs was his risk factor, inquired if contact with infected blood on cuts of the skin could lead to infection. He said that in 1982–88 he worked as a truck driver, primarily in the New York/New Jersey area. During that time he admitted that he and work colleagues would go out "gay bashing". They sought out places frequented by gay men and systematically beat them. The patient admitted doing this "too many times to count", and would frequently get large amounts of the victim's blood on himself. He frequently sustained small lacerations on his hands while administering the beatings. On the patient's first examination we noted several small scabbed lacerations over his proximal interphalangeal joints that had, allegedly, resulted from a recent fight.

Mucocutaneous exposure to infected blood has been confirmed as the route of infection in several health-care workers with AIDS.¹ One man seroconverted after contact with another's blood when they both sustained numerous lacerations in a motor vehicle accident.² Although we cannot prove that contact with blood from infected persons during gay bashing was the mechanism of acquisition in this case, it is certainly plausible. Perhaps this case will serve as a deterrent to the dreadful practice of "bashing" people simply because they belong to a particular minority.

Department of Internal Medicine,
Medical Center,
University of Nebraska,
Omaha, Nebraska 68198, USA

PAUL CARSON
JONATHAN C. GOLDSMITH

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Transiently positive HIV antibody test after treatment with tetanus immune globulin

SIR,—A 25-year-old nurse injured her hand on the edge of an operating-table. She was given a prophylactic injection of tetanus immune globulin (TIG). The following day a blood sample was taken to test for hepatitis B virus (HBV) markers and HIV antibody, as is routine at this hospital in cases of possible accidental exposure to HBV and HIV. HBV markers were negative but she was anti-HIV positive with both ELISA tests used in our laboratory (Sorin Biomedica, Behring), and the same sample was western blot positive (Sorin Biomedica), showing reactivity against all viral proteins. A careful history excluded any risk factor for HIV infection other than hospital employment. A second blood sample, taken 12 days later, was anti-HIV negative by ELISA but showed reactivity for gp120 and p24 by western blot. A third sample, taken 1 month after the first, was negative by both ELISA and western blot.

The passive transfer of HIV antibodies by hepatitis B immune globulin (HBIG) has been demonstrated^{1–4} but neither HIV nor

HIV proteins have been detected in HBIG. The history and serological findings in this case suggest passive transfer of HIV antibodies by TIG given 24 hours before the first blood sample was taken. This could explain the initial reactivity, and the progressive disappearance of reactivity in western blot tests is consistent with this explanation. We could not test the TIG preparation for HIV antibody because neither lot number nor manufacturer were known.

A. GONNELLI
P. ALMI
M. RUBINO
M. TOTI

Division of Infectious Diseases,
Ospedale S. Maria della Scale,
53100 Siena, Italy

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Potential molecular competitor for HIV

SIR,—The theory of genotypic selection (ref 1 and unpublished) predicts that a non-pathogenic HIV-1 strain might be identified that could compete in vivo with virulent strains and ameliorate the course of disease.

The phenotype of a non-pathogenic virus (which may be confused with "long-latency") should result in a symptom-free HIV-positive status. An epidemiological search of one affected population identified an individual who might be harbouring such a strain: he had HIV-1 (DNA-PCR *gag*, strong western blot, probable infection for longer than 10 years, no antiviral treatment), T4 lymphocyte counts about 1000/ μ l, and normal mitogenic and delayed-type hypersensitivity skin test (DTH) responses. History revealed multiple high-risk contacts and intravenous drug abuse, suggesting exposure to numerous wild-type pathogenic strains from partners who subsequently died from AIDS as early as 1983. Viral growth in culture was uncharacteristically slow. This donor was negative for active hepatitis, syphilis, herpes, cytomegalovirus, Epstein-Barr virus, toxoplasma, cryptococcus, and histoplasma.

11 severely immunocompromised patients (T4 mean 66/ μ l) with immunological deterioration and disease progression despite zidovudine or dideoxyinosine treatment provided informed consent and agreed to discontinue all antiviral treatment. Each patient received two inoculations of blood (0.5–2.0 ml) from the putative non-pathogenic donor. Strains from symptomless donors must be characterised for competition and non-pathogenicity before inoculation. Patients have now been followed up weekly for six months and clinically 4 have improved, 3 have shown a mixed response, 3 have regressed, and 1 has died. Apart from transient myalgia, low-grade fever, and diarrhoea, no new symptoms were noted. After a zero to trace DTH response at baseline, all 10 survivors progressed to a moderate to strong response in six months (one responding to all of eight antigens and another to seven of eight), suggesting a gradual return of cell-mediated immunity.* Average total T, T4, and T8 cell numbers and percentages remained stable, fluctuating around baseline mean for six months. The above observations are paradoxical in the absence of antiviral therapy or in the presence of progressive immunological deterioration, but indicate that no harm was caused by the infusions.

Importantly, an epitope of p17 (a fragment of *gag* product), appearing strongly in the donor strain and initially absent or weakly present in the inoculated population, subsequently increased strikingly in all patients who improved.

These preliminary findings suggest a variable degree of in vivo colonisation and perhaps competition between a putative non-pathogenic strain and wild-type virulent strains. If the inoculated strain merely had a phenotype of long latency and pathogenicity, we should not have seen clinical improvement and restoration of

*Details available from *The Lancet* or Immuvax.