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Infect Control Hosp Epidemiol 2006; 27:99-101

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Effectiveness of Hepatitis B Vaccination

TO THE EDITOR—The theoretical basis for the effectiveness of hepatitis B (HB) vaccination for the prevention of HB virus (HBV) infection has long been accepted; however, to our knowledge, no cases have been reported in which the effectiveness could be conclusively shown in a single human. Recently, a 50-sample pooled nucleic acid amplification test conducted at the Kanagawa Red Cross Blood Center identified an HBV-positive donor (a 43-year-old male public official) who was suspected to be in the HBV window period. His serum HBV DNA level was found to be 6,300 copies/mL by single-sample nucleic acid amplification test; the infection was with a genotype B strain. The donor was positive for anti–HB surface antigen antibody (anti-HBs), but the titer was relatively low (35 mIU/mL) (EIA cutoff value, 5 mIU/mL). Findings for anti–HB core antibody (anti-HBc), HB surface antigen, HB e antigen, and anti–HB e antigen antibody were all negative. The donor kindly provided a second blood sample, and, curiously, the HBV DNA had disappeared—16 days after the first blood donation, the anti-HBs titer had rapidly increased to 574 mIU/mL, and a test for anti-HBc IgM yielded positive results. A confirmation test conducted 30 days after the first donation showed the same results—the donor was negative for HBV DNA and was positive for both anti-HBs (titer, 383 mIU/mL) and anti-HBc IgM. The donor's alanine aminotransferase level was less than 20 IU/L throughout the month he was followed. Testing of plasma from a preserved donation made 372 days earlier showed that, at that time, he was negative for all HBV markers, even anti-HBs (titer, 0.1 mIU/mL). The seroconversion to anti-HBc IgM was deemed to be conclusive evidence of first-time HBV infection. The infection route was not apparent, because he reported no sexual activity or needlestick injury. The donor had received HB vaccination 20 years earlier (at 23 years old), when he worked at a rehabilitation hospital. We concluded that, because of the HB vaccination, the transmitted HBV was cleared rapidly by a secondary immune response.

This case supports the view that HB vaccination is effective against HBV transmission in immunocompetent individuals and also shows that memory cells produced in response to vaccination can last for more than 20 years,[1] even if the anti-HBs titer is very low. To our knowledge, this is the first successful documentation of the effectiveness of HB vaccination in a single human. We were fortunate to identify, from 20 million blood donors in Japan, an individual in the HBV window period who had previously been vaccinated and for whom blood had been stored from a previous donation. Although this very short transient viremia was probably a standard immune reaction to HB vaccination at the DNA level, it brings up another problem for blood centers. Even though a donor has been vaccinated, a short period of viremia may occur after exposure to HBV, which would result in a high risk of HBV transmission by transfusion. Furthermore, it is important that the question of whether such donors should be allowed to reenter donor programs after viral clearance be discussed.

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Prevalence of and Risk Factors for Colonization With Vancomycin-Resistant Enterococcus Among Human Immunodeficiency Virus–Positive Outpatients

TO THE EDITOR—Patients infected with human immunodeficiency virus (HIV) are at increased risk for infection with both typical community-acquired pathogens and drug-resistant organisms. HIV-positive patients often receive antibiotic therapy and have frequent contact with the healthcare system, both of which are factors that have been associated with an increased risk of infection with vancomycin-resistant enterococcus (VRE) in other populations.[1,2] One study of hospitalized patients with enterococcal bacteremia showed that they were more likely to be infected with vancomycin-resistant strains if they were HIV positive or had a history of AIDS.[1]

In spite of the clinical importance of colonization as a precursor to infection with drug-resistant pathogens,[2,3] little is known about the frequency with which HIV-positive outpatients are colonized with VRE. Moreover, specific risk factors for carriage have not been elucidated in this population. The present study was performed to determine the prevalence of VRE colonization among HIV-positive patients visiting an ambulatory clinic. The objectives were to assess the overall percentage of VRE carriage, as determined by analysis of perirectal swab specimens, and to characterize patients who may be at increased risk of VRE colonization.

After approval by the Institutional Review Board, the study was performed at the University of Chicago Infectious Diseases Clinic (Chicago, IL). From November 2003 through April 2004, HIV-positive patients seen during routine care at the clinic were considered for inclusion in the study. For patients who provided consent, a perirectal swab specimen was obtained using the Sterile BBL CultureSwab Plus collection system (Becton, Dickinson, and Company). The swab was dipped into sterile water, applied to the anal verge, and placed into transport culture media. Because colonization status was not known prior to collection of the swab specimens, no additional measures beyond standard precautions were followed in the care of these patients. Multiple swab specimens were obtained during a clinic session, and all of them were transported to the microbiology laboratory together. Swabs were inoculated on enterococcosel plates and were examined daily. Colonies that were morphologically suggestive of enterococcal species underwent biochemical examination to confirm identity, and susceptibility to vancomycin was confirmed by use of Etest (AB Biodisk). Swab results were made available to healthcare providers. Patients were notified of their carriage status at the time of the subsequent clinic visit. VRE isolates were incubated on blood agar plates and were stored at −70°C in a freezer until genotypic analysis was performed. After subculturing twice to blood agar plates, genomic DNA was extracted, digested using *Sma*I, and subjected to pulsed-field gel electrophoresis.[4,5] Images of band patterns were acquired by use of the GelDoc2000 system (BioRad) and were compared with each other, to ascertain relatedness, by use of the Bionumerics interpretation software package (Applied Maths).

After the swab specimen was obtained, the patient's information (ie, demographic characteristics, dates of recent hospitalization, medication use, and immunologic and virologic status) was extracted from the clinic chart and electronic medical records. Comparison of patient characteristics was performed using the Wilcoxon rank sum test for continuous variables and Fischer's exact test for dichotomous variables.

Eighty-five HIV-positive patients provided consent and were included in this study (Table). Four (4.7%) of the 85 patients studied were found to be colonized with VRE. None of the colonized patients demonstrated signs or symptoms of active infection with VRE. In comparison with VRE-negative control patients, colonized patients were significantly more likely to be receiving trimethoprim-sulfamethoxazole prophylaxis ($P = .05$), to have a lower nadir CD4 cell count (median, 20 vs 166 cells/mm^3; $P = .05$), to have a lower recent CD4 cell count (median, 136 vs 401 cells/mm^3; $P = .02$), and to have been hospitalized more recently (median, 68 vs 440 days since last hospitalization; $P = .02$). Results of pulsed-field gel electrophoresis demonstrated that the 4 VRE isolates were not genetically related.

The prevalence of colonization with VRE in the population studied was low but was higher than reported elsewhere among HIV-positive outpatients. In an earlier study of fecal VRE carriage in 89 HIV-positive outpatients, none was found to be colonized with VRE.[6] The prevalence of VRE colonization observed in the present study is comparable to that observed for methicillin-resistant *Staphylococcus aureus* in HIV-positive outpatients in 2 earlier studies.[7,8] In addition, the level of VRE colonization described here is similar to those reported for other high-risk populations, such as patients awaiting solid-organ transplant.[9]

The risk factors for VRE colonization suggested here are also similar to those reported elsewhere for other outpatient groups colonized with drug-resistant pathogens. Carriage of drug-resistant bacteria has been consistently shown to be more likely among patients with extensive exposure to an-