Incidence of Human Immunodeficiency Virus Antibody in a Prenatal Population at a Community Hospital

THOMAS S. ALEXANDER,1,2* JOANNE LEE,2 AND BELINDA YEN-LIEBERMAN3

Department of Pathology and Laboratory Medicine, Summa Health System, Akron, Ohio 44304; Northeastern Universities College of Medicine, Rootstown, Ohio 44272; and Department of Laboratory Medicine, Cleveland Clinic Foundation, Cleveland, Ohio 44106

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Prenatal human immunodeficiency virus (HIV) screening may reduce vertical HIV transmission. We screened 4,419 prenatal sera and found 38 repeatedly reactive specimens with an HIV-1–HIV-2 enzyme-linked immunosorbent assay. Western blot analysis confirmed four of these specimens as positive for HIV-1 antibodies. Screening detects previously unidentified HIV infections, but false-positive results may also occur.

The Pediatric AIDS Clinical Trials Group (PACTG 076) demonstrated that human immunodeficiency virus (HIV) transmission from mother to infant can be reduced by 67.5% in women with HIV infection and CD4 counts of ≥200/mm3 by treating the mother with zidovudine during pregnancy (2). These results have led to proposals to require mandatory screening of pregnant women or newborns for HIV (10) in order to reduce vertical transmission. In fact, newborn screening has been performed (3, 6) and is currently under way in the state of New York. Screening of infants does not reduce vertical transmission for two reasons. First, the HIV antibody assay detects immunoglobulin G antibody that crosses the placenta. Therefore, any infant born to an HIV-infected mother will test positive in an HIV antibody test regardless of its infection status. Indeed, such screening has been the cornerstone of attempts by the Centers for Disease Control and Prevention (CDC) to determine seroprevalence in pregnant women (3). Second, reduction in vertical transmission has been associated with treatment of the mother before delivery (2), and this would not be possible if the antibody testing was performed on the newborn. Postbirth PCR screening of high-risk newborns could identify HIV-infected infants who might be helped by immediate antiretroviral treatment (1). Both mandatory and voluntary HIV antibody screening of pregnant women has been proposed (10), with social, legal, and medical arguments for and against each proposal (4). An essential issue is the prevalence of HIV in pregnant women and whether testing will provide accurate results in that population. Indeed, multiparity has been reported to be a cause of false-positive enzyme-linked immunosorbent assay (ELISA) results (9). The CDC has presented HIV prevalence data from 1993 ranging from 0 to 0.57% in pregnant women depending on the women’s state of residence (10). The CDC reported an HIV-1 seropositivity rate of 0.06% for pregnant women in Ohio during 1993 (10). Ohio has not enacted any legislation that addresses HIV testing in pregnant women. Although studies have documented the seroprevalence of HIV antibodies in pregnant women in areas of endemicity in Kigali, Rwanda (7), Rakai (11), and England (5), published data on HIV seroprevalence documented by maternal testing in the United States are lacking. The New York State Department of Health has published a pamphlet documenting HIV seroprevalence data for childbearing women obtained by newborn testing and has shown an overall positivity rate of 0.43% for 1996 (8).

We determined the incidence of HIV antibodies in sera submitted for prenatal testing to a large community teaching hospital in Akron, Ohio. The population consisted of women presenting to either private physicians or the hospital prenatal clinic for prenatal screening. We obtained Summa Health System Institutional Review Board approval to encode 4,419 sera which had been submitted to our laboratory for rubella antibody testing from 1993 to 1996. Ohio law prohibits HIV antibody testing without written informed consent if the results may be linked to patient identification in any manner. Thus, no demographic data are available on our population except that it consisted of females presenting for prenatal testing. The encoded specimens were tested for antibodies to HIV by using the Sanofi Diagnostics/Genetic Systems HIV-1–HIV-2 combination viral lysate ELISA (Sanofi Diagnostics, Redmond, Wash.). All reactive specimens were retested in duplicate. Repeatedly reactive specimens, defined as initially reactive specimens which were reactive on one or both replicates, were assayed by HIV-1-specific Western blotting (Organon Teknika, Durham, N.C.).

We found 38 specimens (0.85%) to be repeatedly reactive by ELISA; four of them (0.09%) contained bands to at least p24, gp41, p51, gp120, and gp160. These four specimens met criteria defined by the Association of State and Territorial Public Health Lab Directors, CDC, the American Red Cross, and the Consortium for Retrovirus Serology Standardization for being called Western blot positive (9). Fourteen of the ELISA repeatedly reactive specimens (0.31% of total) produced bands that did not meet the positive criteria; these specimens were classified as Western Blot indeterminate. Six of the Western blot-indeterminate specimens had a p18 band only, two had a p24 band, and another had a nondiagnostic band, while five specimens were read as borderline for either a p18 or a p24 band (Fig. 1). The remaining 20 ELISA-reactive specimens (0.45% of total) were negative by the Western blot procedure. We had sufficient serum to test nine of the Western blot-indeterminate specimens and 11 of the Western blot-negative specimens for HIV-1 p24 antigen. All 20 specimens were negative for HIV-1 p24 by the Organon Teknika Base Dissociated p24 ELISA procedure.

Our incidence of confirmed positives (0.09%), while low, is 50% higher than the CDC estimate (0.06%) for the state of Ohio (10). We cannot determine if this difference is statistically
can be expected. Thus, an appropriate confirmatory test, such as a Western blot or immunofluorescence assay, must be included in the algorithm. Appropriate followup testing of women with negative or indeterminate Western blot results depends on clinical and demographic data for individual patients. Such tests might include HIV-1 PCR, repeat antibody testing, or HIV-2-specific testing, depending on the patient’s clinical history.

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REFERENCES


