SHORT COMMUNICATION

Toxoplasma gondii Antibodies in HIV and Non-HIV Infected Thai Pregnant Women

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HIV infected pregnant individuals are susceptible to variety of infections including toxoplasmosis.¹ The potential of the intracellular protozoan parasite, Toxoplasma gondii, to cause severe complications has been documented in pregnant women and the immunologically impaired. Transplacental transmission may be a cause of abortion, stillbirth, and serious congenital anomalies.² The most frequently observed clinical manifestation in symptomatic patients is lymphadenitis which may be associated with fever, fatigue, muscle pain, sore throat, and headache. The symptoms usually subside spontaneously in weeks or months.³ In the compromised host, T. gondii has been recognized as an important opportunistic pathogen causing fulminant infection including necrotizing encephalitis, diffuse encephalopathy, cerebral mass lesion, pneumonitis, myocarditis and hepatitis.⁴,⁵ In approximately 90% of immunocompetent individuals, no clinical symptoms of toxoplasmosis are apparent during acquired acute infection. However, even in an asymptomatic newborn, developmental defects can appear later in life causing blindness, hearing loss and mental retardation.⁶ Prenatal diagnosis of toxoplasmosis and antiparasitic treatment during pregnancy may prevent fetal infection and organ damage.

In Thailand, the incidence of HIV infection in the obstetric population has been reported to be 2.4%,⁷ but data regarding the prevalence of Toxoplasma infection in HIV-seropositive pregnancies are limited. According to Chintana et al.,⁸ the seroevidence of IgG to T. gondii in 19 and 1,181 pregnant women with and without HIV infection was 21.1% and 13.1%, respectively. The significant difference between the groups could not be evaluated because the summary

SUMMARY Serological evidence for Toxoplasma gondii infection in Thai pregnant women was investigated. One thousand six hundred and sixty-nine blood specimens were collected from 838 HIV-seropositive and 831 HIV-seronegative pregnant women attending the antenatal-care clinic at Siriraj Hospital, Bangkok, Thailand, during a two-year period. Toxoplasma IgG antibody was detected, using a solid-phase enzyme-linked immunosorbent assay in which the membrane protein p-30 was the predominant antigen. IgG positive sera were subsequently examined for IgM antibody by the capture antibody enzyme immunoassay. The IgG antibody was found in 450 (53.7%) HIV seropositive women and 44 (5.3%) non-HIV infected women, with a statistically significant difference (p < 0.0001). Three of the 450 HIV-seropositive and 2 of the 44 HIV-seronegative sera with IgG antibody were positive for IgM antibody against T. gondii. This result suggested that HIV seropositive pregnant women had a higher risk of Toxoplasma infection with increase exposure to their offspring.
insufficient number of the HIV-seropositive group. The aim of this study was to evaluate the infection with toxoplasmosis in HIV and non-HIV infected Thai pregnant women.

MATERIALS AND METHODS

The size of the study populations was calculated by the SAM program. One thousand, six hundred and sixty-nine blood specimens were randomly collected from 838 HIV-seropositive and 831 HIV-seronegative pregnant women who visited the antenatal-care clinic at Siriraj Hospital, Bangkok, Thailand, from 1997 to 1999. Toxoplasma IgG antibody was detected by the solid-phase enzyme immunoassay technique (Platelia® Toxo IgG, Sanofi Diagnostics Pasteur, France) in which the membrane protein p-30 was the high predominant antigen. Informed consent was obtained from all participants after the purpose and nature of this study were explained to them.

Sera positive for the IgG antibody were further evaluated for IgM antibody to Toxoplasma by the capture antibody enzyme immunoassay method (Platelia® Toxo IgM, Sanofi Diagnostics Pasteur, France). The test utilized a monoclonal antibody to T. gondii labeled with horseradish peroxidase for enzyme immunoassay signal development. The Chi-square test was used for statistical analysis.

RESULTS

Four hundred and fifty (53.7%) out of 838 HIV infected and 44 (5.3%) out of 831 non-HIV infected pregnant women were positive for T. gondii IgG antibody. The difference in seroprevalence between the two groups was statistically significant (p < 0.0001). Only 3 HIV-seropositive and 2 HIV-seronegative parturients were found to be positive for T. gondii IgM antibody.

Of the cases with positive Toxoplasma IgM antibody, two of the 3 HIV infected women had an elective abortion. The other HIV-seropositive women and one HIV-uninfected case were lost to follow-up. Only one non-HIV infected mother-child pair with positive IgM mother could be evaluated after parturition; the mother was asymptomatic and the infant was born healthy. At six months, the infant developed hydrocephalus. A combination of pyrimethamine, sulfadiazine and folinic acid was prescribed for 2 months. At the next two visits, the child's symptoms did not progress. The baby was finally lost to follow-up.

DISCUSSION

Since the importance of toxoplasmosis as an opportunistic infection in AIDS patients has been considered, there appears to be a growing number of reports describing the epidemiology of Toxoplasma in pregnant women with HIV infection. Primary maternal T. gondii infection during pregnancy may be associated with significant morbidity and mortality in the newborns. The possibility of congenital toxoplasmosis in infants born to mothers dually infected with Toxoplasma and HIV may be higher than those born to mothers infected with Toxoplasma alone. The risk of congenital toxoplasmosis has encouraged many researchers to study the seroepidemiology of T. gondii antibodies in pregnant women.

This study showed that the seroprevalence of Toxoplasma infection in HIV infected pregnant women was significantly higher than that in HIV-seronegative pregnant women (53.7% vs 5.3%, p < 0.0001). Our data was in concordance with the study of Chintana et al. in which IgG antibody rates to T. gondii were found to be higher in HIV-seropositive than in HIV-seronegative Thai pregnant women (21.1% vs 13.1%). However, the number of HIV-seropositive cases was too low to evaluate whether the difference was statistically significant. Comparable data from Tanzania demonstrated a tendency towards a higher rate of T. gondii seropositivity among HIV infected pregnant women.

Presence of Toxoplasma IgG antibody indicates previous infection and possible existence of cysts within the tissues. In immunocompromised patients, rupture of
a tissue cyst may result in liberation of bradyzoites and regeneration of parasites. The secondary reactivation of a previous chronic or quiescent Toxoplasma infestation in those with declining immunity might be an explanation of a higher seroprevalence among HIV infected pregnancies. This information implied a greater risk of maternal-fetal transmission of Toxoplasma in pregnant women who were dually infected with both agents.

Our data showed that the similar numbers of subjects who were positive for Toxoplasma IgM antibody were found in both groups, which implied that the acute infection rate of Toxoplasma in HIV infected pregnancies was not different from that in non-HIV group. This supports a previous study which suggested that most of the infection occurred long before pregnancy in Thai women. Because congenital toxoplasmosis can occur in children born to mothers with latent Toxoplasma infection if they are HIV infected, the high Toxoplasma seroprevalence among HIV seropositive pregnant women poses a potentially high risk for their babies. The data in this study underscored the importance of toxoplasmosis in the Thai pregnant population.

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REFERENCES