AIDS briefs

Trichomonas and HIV infection

Infection with *Trichomonas vaginalis* significantly increases a woman's risk of becoming infected with HIV, according to a study published in a recent edition of the *Journal of Infectious Diseases*.

It is well known that sexually transmitted infections (STIs) increase the risk of transmitting and acquiring HIV. Researchers from the University of Washington, USA, and from Mombasa, Kenya, thought there might be a link between *T. vaginalis* infection and an increased risk of acquiring HIV and carried out an 11-year prospective study of sex workers in Mombasa, running between 1993 and 2004. The study involved 1 335 female sex workers who were HIV negative at baseline. The women were seen monthly, when they provided information about their general medical, gynaecological and sexual health. They also had physical examinations and were screened for HIV, as well as other STIs, including *T. vaginalis*.

During the study, researchers recorded 806 infections with *T. vaginalis* and 265 women became infected with HIV. Infection with *T. vaginalis* significantly increased the risk of HIV infection, even after adjusting for possible confounding factors. *T. vaginalis* infection itself was significantly associated with a shorter duration of sex work, less than 8 years of education and concurrent cervicitis and bacterial vaginosis. Women who used condoms 100% of the time, as well as those who used progesterone-only contraceptives, had a lower risk of *T. vaginalis* infection.

Researchers suggest several possible reasons why infection with *T. vaginalis* increases the risk of HIV acquisition:

- *T. vaginalis* leads to inflammation, resulting in cells vulnerable to HIV infection being present in the vaginal and cervical mucosa.
- *T. vaginalis* could cause mucosal haemorrhage, damaging natural defences against infection.
- *T. vaginalis* undermines a process that can prevent HIV's attachment to cells.
- *T. vaginalis* increases the risk of HIV by increasing susceptibility to vaginal infections or the persistence of abnormal vaginal flora.

McClelland RS, *et al. J Infect Dis* 2007;195: 698-702 (online edition).

Acute HIV infection may be masked in testing

Almost 1 in 40 of those who tested HIV negative in a large clinic cohort in Lilongwe, Malawi, turned out to have acute HIV infection that was too recent to be detected by single or dual rapid antibody test used as the standard method of HIV diagnosis, researchers from the University of North Carolina report in a recent edition of the *Journal of Infectious Diseases*.

Researchers suggest that the sensitivity of the test could be improved by using a second rapid test alongside the first one in all patients and not just in those who have indeterminate results or who test HIV positive. The alternative would be a p24 antigen test in the case of indeterminate results. Antigen testing is cheaper and easier to carry out than pooled HIV RNA testing.

Researchers think that their results confirm that in areas of high HIV prevalence, the sensitivity of a single HIV antibody test can be as low as 96% because of the presence of significant numbers of previously unrecognised acute HIV infections in the population being tested.

These findings are important because they show that in an African sexually transmitted disease clinic cohort, up to 4% of patients tested for HIV during a 1-year period were experiencing acute HIV infection but had not yet begun to produce antibodies, suggesting that current HIV testing methods in African settings could be missing a substantial number of highly infectious individuals. During acute HIV infection HIV transmission is much more likely than during the chronic phase of infection because virus levels in genital fluids and blood are very high.

Researchers screened 1 450 patients presenting with sexually transmitted infections over the course of 21 months at Kamuzu Central Hospital in Lilongwe, Malawi.

Antibody testing was carried out according to the Malawian national protocol, which requires the use of two rapid tests (the Abbott Determine and the Trinity Biotech Unigold tests). Patients with concordant positive results were diagnosed as HIV positive immediately, while patients with concordant negative results were asked to return 1 week later to receive the result of a confirmatory test. Patients with discordant results were also asked to return 1 week later.

All negative and discordant samples were screened using HIV RNA and HIV p24 antigen tests, and Western blot testing was carried out on all samples where HIV RNA was detected.

Twenty-one cases of acute infection were identified by HIV RNA testing. In comparison, ultrasensitive p24 antigen testing identified 15 of 17 (88% sensitivity), standard p24 antigen testing identified 12 of 16 (75% sensitivity) and discordant rapid antibody tests identified 7 of 21 (33% sensitivity). This amounted to a prevalence of acute HIV infection of 2.4% among initially seronegative patients.

The authors concluded that in a clinic population of 10 000 patients treated per year, the use of concordant rapid tests to detect HIV would miss around 145 cases of acute HIV infection and 14 cases of established HIV infection. They also said that 97 people would have a false positive HIV test. If an ultrasensitive p24 assay were used, only 16 of 132 cases of acute HIV infection would be missed.

Fiscus SA, *et al. J Infect Dis* 2007; 195: 416-424.

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