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Abstract

Associations between herpes simplex virus type-2 (HSV-2) and bacterial vaginosis (BV) in a cohort of HIV-1 infected women in Burkina Faso

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Objectives Observed associations between herpes simplex virus type 2 (HSV-2) and bacterial vaginosis (BV) remain poorly understood. We describe the relationships between HSV-2 and BV among HIV-1 infected women not needing highly active antiretroviral therapy in Burkina Faso.

Methods 136 HSV-2 and HIV-1 seropositive women were randomised in a double-blind placebo-controlled trial of HSV suppressive therapy. Genital samples were collected at 12 visits over 3 months (including 6 visits before initiating study drugs), and analysed for HSV-2 DNA shedding (real-time PCR) and vaginal flora (Nugent's score). Associations between genital HSV-2 DNA and BV were investigated using generalized estimating equations logistic regression.

Results Among the 68 women in the placebo arm, the prevalence of HSV-2 DNA genital shedding and BV over 12 visits were 15.2% (116/761) and 29.1% (222/774), respectively. Genital HSV-2 DNA was associated with an increased prevalence of BV when detected at the concomitant visit (41.4% in presence of shedding vs. 26.7% in absence, OR=1.58, 95% CI 1.13–2.21) or at the previous visit (38.8% in presence of shedding vs. 26.6% in absence, OR=1.35, 95% CI 0.88–2.07). Similarly, genital HSV-2 DNA was more likely to be detected if BV was present at the previous visit (21.1% if BV present vs. 13.4% if absent, OR=1.40, 95% CI 0.87–2.25).

However, prescription of metronidazole (for BV or *Trichomonas vaginalis*) was not associated with a reduced risk of genital HSV-2 DNA at the subsequent visit (OR=1.03, 95% CI 0.65–1.62); and analyses including all 136 women randomised to valacyclovir or placebo found no impact of HSV-2 treatment on BV occurrence (OR=0.86, 95% CI 0.58–1.28).

Conclusions BV and HSV-2 genital shedding were associated in this population of HIV-1 infected women, but we did not find any effect of HSV-2 or BV treatment. Further research is needed to elucidate the possible mechanisms by which the infections might interact.