018.5

FALSE NEGATIVE HSV IGG1 AND IGG2 ANTIBODY RESPONSES IN INDIVIDUALS WITH A RECURRENT GENITAL HERPES INFECTION

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Introduction HSV type specific antibodies (IgG1 and IgG2) are produced and persist after HSV-1 and –2 infections. Therefore, IgG2 HSV serologic tests are used as an indicator for genital herpes history (e.g. in pregnant women), to counsel partners of genital herpes patients, and as a proxy marker of high-risk sexual behaviour. We tested this paradigm by retrospectively measuring IgG1 or IgG2 HSV antibodies in sequential serum samples from visitors with symptomatic PCR proven recurring genital herpes episodes.

Methods We selected individuals with two episodes of PCR proven HSV-1 or HSV-2 genital herpes, which were at least 3 months apart. Serum samples collected during the second (recurring) episode were tested with a HSV type specific ELISA and Immunoblot (both Focus HerpeSelect®) for anti-gG1 or anti-gG2 antibodies. The immunoblot was used as a reference test.

Results From May 2006 to December 2010 we selected 18 and 35 individuals with recurrent HSV-1 or HSV-2 genital herpes, respectively. In the HSV-1 cohort, serum sample testing showed that 13 out of 18 (72%) samples were tested positive in both tests, 2 out of 18 (11%) samples were positive in the gG1 ELISA but negative in the immunoblot, and 3 out of 18 (17%) samples were negative/equivocal in both tests. In the HSV-2 cohort, serum sample testing showed that 29 out of 35 (83%) samples were positive in both tests, 1 out of 35 (3%) was positive in the gG2 ELISA but negative in the immunoblot, and 5 out of 35 (14%) samples were negative in both tests.

Discussion Our data show that HSV IgG1 and IgG2 antibodies are false negative in respectively 28% and 17% of recurrent genital infections. This should be taken in consideration when these tests are used in a clinical setting or as proxy for risk behaviour in epidemiologic studies.

018.6

PERSISTENCE OF PHARYNGEAL CHLAMYDIA TRACHOMATIS FOR 1–2 WEEKS IS COMMON AMONG CLIENTS AT THE AMSTERDAM STI CLINIC

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Background Pharyngeal *Chlamydia trachomatis* (PCt) must persist to contribute to ongoing transmission. We examined the prevalence, persistence and determinants of PCt among STI clinic clients. **Methods** All men having sex with men (MSM) and women with a high risk profile were screened for anogenital and pharyngeal Ct with the APTIMA *Combo* 2 assay. After one week, clients with PCt were recalled for treatment, a follow-up pharyngeal swab and a questionnaire. Clients who used antibiotics since first visit were excluded from the analysis.

Results Between January 2011 and July 2012, we detected 148 PCt in MSM (13,111 visits; prevalence 1.1%) and 160 PCt in women (6,915 visits; 2.3%). In both groups, PCt was associated with being

notified for STI, concurrent urogenital Ct and > 10 partners, but not with pharyngeal symptoms. Women reporting sex work had a lower risk, while women with pharyngeal gonorrhoea and MSM with anorectal Ct had a higher risk for PCt. 53% of MSM and 32% of women with PCt had no concurrent anogenitial Ct.

Of 43 (29%) MSM and 55 (34%) women, follow-up swabs and questionnaire data were available. The median time between both visits was 10 days. PCt persisted in 27 (63%) MSM and in 35 (64%) women. In both groups 50% had unprotected active oral sex between first and second visit, but this did not affect persistence.

Among MSM no determinants for persistence were detected; among women being notified for STI, younger age and urogenital Ct were significantly associated with persistence in univariate analysis.

Among clients (n = 16) whose second visit was more than 3 weeks after the first visit, 11 (69%) had PCt at second visit.

Discussion The prevalence of PCt is low among STI clinic clients, but persistence is common. Therefore, the pharynx is a potential reservoir for ongoing Ct transmission.

0.19 - Prevention and curing STIs: Who is the winner?

019.1

AZITHROMYCIN AND DOXYCYCLINE RESISTANCE PROFILES OF MYCOPLASMA GENITALIUM AND ASSOCIATION WITH TREATMENT OUTCOMES

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Background Antibiotic resistance profiles of recent *Mycoplasma genitalium* (MG) isolates have not been extensively evaluated nor correlated with treatment outcomes for NGU. Urine specimens from men with NGU enrolled in a treatment trial in Seattle, WA were used to culture MG strains and assess the association of their susceptibility to azithromycin and doxycycline with treatment outcomes. **Methods** Urines from all MG-positive (by PCR) men were co-cultured with VERO cells. MG growth was detected by an increase in genomes using an MG-specific quantitative PCR (qPCR); minimum inhibitory concentrations (MICs) were defined by the antibiotic concentration that resulted in 99% growth inhibition. MICs were measured at baseline (V1), 3-week (V2) and 6-week follow-up (V3). Clinical cure (V2, V3) was defined < 5 PMNs/HPF, no urethral discharge or symptoms; microbiologic cure was defined by a negative MG-specific PCR result.

Results Viable MG strains were recovered from 141 (92%) of 153 MG PCR-positive specimens; MICs were determined on 103 isolates. Azithromycin MICs were clearly bimodal; 46% (48/103) were ≤ 0.001–0.5 μ g/ml, considered susceptible, and 54% (55/103) were ≥ 8 μ g/ml considered resistant. Except for two strains with MICs of ≥ 8 μ g/ml doxycycline, MICs were < 0.125–2 μ g/ml. Doxycycline MICs did not correlate with treatment outcomes. At baseline, 33/57 (57.9%) of isolates had azithromycin MICs that were resistant. Of men in the azithromycin arm with MIC data and treatment outcomes at V2, 11/13 clinical failures (84.6%) and 16/20 microbiologic failures (80.0%) had azithromycin resistant isolates at baseline. After receiving azithromycin, 9/10 V2 clinical failures (90%) and 12/13 microbiologic failures (92.3%) had V2 azithromycin resistant MICs. All V3 clinical (7/7) and microbiologic (10/10) failures had V3 azithromycin resistant MICs.

Conclusion Approximately 60% of MG strains were resistant to azithromycin at baseline; azithromycin treatment failures occurred in 90–100% of men who received azithromycin. Development of new antimicrobial therapies for MG is essential.