DETERMINING THE ORIGINS OF REPEAT ASSESSMENT OF URETHRITIS ETIOLOGY AMONG HIV-INFECTED MEN IN LILONGWE, MALAWI

Background Malawi uses syndromic management for the treatment of sexually transmitted infections (STIs). However, the etiology profile of STIs has been shown to change over time. We conducted a current assessment for etiology of urethral discharge (UD) among men in Lilongwe Malawi to inform development of effective national treatment guidelines.

Methods We enrolled a cohort of HIV infected men with UD who were either ART naïve or on ART for ≥12 weeks at Bwaila STI clinic. We collected blood samples and urethral swabs for STI etiologic testing as follows: Neisseria gonorrhoeae (GeneXpert, culture), Chlamydia trachomatis (GeneXpert, culture), and Trichomonas vaginalis (OSOM – Trichomonas Rapid Test). All patients were treated syndromically with gentamicin, doxycycline, and metronidazole, per Malawian standard of care.

Results Of 189 men enrolled between January 1, 2017 and December 31, 2018; 87 (46.0%) were not on ART, and 102 (54.0%) were on ART. Participants reported urethral discharge for a median of 4 days (IQR: 3, 7). Among participants, 152 (80.4%) tested positive for gonorrhea via GeneXpert and 124 (66.7%) via culture; 17 (9.0%) tested positive for chlamydia, 6 (3.2%) tested positive for trichomonas and 30 (15.9%) did not test positive for any of the three etiologies. 15 (7.9%) participants had multiple STIs. There were no differences in distribution of etiologies (individual or multiple) between men on and not on ART (p≥0.10 for all comparisons).

Disclosure No significant relationships.

P791 ASSESSMENT OF URETHRITIS ETIOLOGY AMONG HIV-INFECTED MEN ATTENDING AN STI CLINIC IN LILONGWE, MALAWI

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Background High rates of repeat T. vaginalis infections post-treatment have been reported. It is essential to understand the origin of these infections (i.e. treatment failure or reinfection) to determine the best secondary prevention measures. Self-reported sexual behavior and medication adherence can be subject to bias. The purpose of this study is to examine the origins of early repeat T. vaginalis infections in women using clinical versus genotype-informed criteria.

Methods Women with T. vaginalis confirmed by culture or nucleic acid amplification test (NAAT), who were randomized to receive 2 g or 7-day 500 mg BID metronidazole (MTZ), were retested 3–12 weeks post treatment at test-of-cure (TOC). Viable isolates from women who were TOC TV+ were genotyped (baseline and TOC isolates) and MTZ susceptibility (TOC only) was evaluated. Sexual and treatment adherence histories were elicited by computer-assisted self-administered survey. Treatment failure was defined using two criteria: 1) clinical (a combination of MTZ adherent per self-report + no follow-up sexual exposure per self-report + no MTZ resistance), and 2) genotype-informed (concordant baseline and TOC genotype with no follow-up sexual exposure per self-report).

Results Of 80 repeat positives, 78 were evaluated using clinical and 49 using genotype-informed criteria. Per clinical criteria, 87.2% were treatment failure, 7.7% were reinfection and 5.1% were new infection. Per genotype-informed criteria, 51.0% were treatment failure, 10.2% were reinfection and 38.8% were new infection. In subset analysis, comparing the 49 with both clinical and genotype-informed assessments, 61.2% agreed and 38.8% disagreed (kappa 0.29 indicating poor reliability). Of 44 women who denied having a new partner during follow-up, 14 (31.8%) had a discordant genotype.

Conclusion Using either criteria, most TOC T. vaginalis positives were treatment failure rather than re-infections. Clinical and genotype-informed classification were not well correlated. Possible explanations for this will be discussed.

Disclosure No significant relationships.

P790 DETERMINING THE ORIGINS OF REPEAT TRICHOMONAS VAGINALIS INFECTIONS USING CLINICAL Versus GENOTYPE-INFORMED CRITERIA

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Background and rationale The data obtained from BD MAX Vaginal Panel runs conducted on 1,740 clinician-collected specimens taken from symptomatic patients was analyzed to determine if any detection patterns emerged. The distribution of BV marker combinations detected in the absence and presence of BV and/or TV were compared.

Results Independent of the BV result (BV+ or BV−), the proportions of samples containing no BV markers and samples containing all BV markers were different in TV− and TV+ samples. TV+/BV− samples displayed a significantly higher number of cases in which only A. vaginae was detected or a combination of A. vaginae and G. vaginalis than in samples found TV−/BV−, TV−/BV+ or TV+/BV+.

Conclusion The BV marker detection patterns vary with the presence of co-infection by TV. The results obtained in this analysis suggest some interplay between BV and TV and warrants further investigation.

Disclosure No significant relationships.