recent partner change and/or self reported vaginal symptoms such as thrush or BV in the preceding 3 months. These are all factors which would potentially impact vaginal microbiota.
Conclusion Pelvic inflammatory disease is known to be associated with sexual behaviour but a sexually transmitted infection is not always detected in PID. Here, we have found preliminary indications that a microbiota previously associated with sexually transmitted infection risk is also associated with PID. This is pilot data and further numbers are needed before conclusions can be reached.
Disclosure No significant relationships.

O05.6 CERVICOVAGINAL METABOLIC PROFILING REVEALS THE INTERPLAY BETWEEN HPV, MICROBIOTA AND INFLAMMATION IN CERVICAL CARCINOGENESIS

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10.1136/sextrans-2019-sti.133

Background Vaginal dysbiosis has emerged as a key risk factor in HPV acquisition, persistence, and potentially cervical carcinogenesis. However, the biological mechanisms driving persistence and carcinogenesis have not been elucidated. Hence, our objective was to perform metabolic profiling of the cervicovaginal microenvironment to identify interactions between virus, host and microbes in the context of genital inflammation, dysplasia, and cancer.
Methods In a multicenter study, metabolic profiles of 78 premenopausal, non-pregnant women with low-grade (LSIL) and high-grade squamous intraepithelial lesions (HSIL), invasive cervical cancer (ICC), or healthy controls (HPV-positive and -negative Ctrl) were analyzed using gas chromatography-mass spectrometry. Metabolome and vaginal microbiome datasets were integrated using state-of-the-art bioinformatic tools (PICRUSt, AMON, and MIMOSA). Hierarchical clustering analysis (HCA) and principal component analysis (PCA) were employed to reveal the influence of genital inflammation, patient groups, and microbiota on metabolic profiles. Receiver Operating Characteristics (ROC) analysis was used to discriminate metabolites for each patient group. Statistical differences were tested using ANOVA or Mann-Whitney U test.
Results Metabolomes of ICC patients (n=468 metabolites) formed a distinct cluster on PCA and HCA plots, due to enrichment of membrane lipids. Amino acid and nucleotide metabolites were depleted in HPV-positive Ctrl, LSIL and HSIL groups ($P<0.05$). Microbial communities were predicted to alter amino acid and nucleotide metabolisms. Eicosenoate, 3-hydroxybutyrate, and oleate/vaccenate (AUC > 0.9, $P<0.01$) discriminated ICC from healthy patients. Sphingolipids and plasmalogens positively correlated with genital inflammation (Spearman's rho > 0.7). Anti-inflammatory nucleotides, adenosine and cytosine positively correlated with *Lactobacillus* abundance (Spearman's rho>0.5) and negatively correlated with genital inflammation (Spearman's rho<-0.3). HCA of metabolites demonstrated that metabolic profiles were driven by cancer, genital inflammation and *Lactobacillus* dominance.
Conclusion The complex virus-host-microbe interplay within the cervicovaginal microenvironment lead to unique metabolic fingerprints that could be exploited for future development of diagnostics, preventatives or treatments to positively impact women's health outcomes.
Disclosure No significant relationships.

O06 – STI/HIV TREATMENT EFFICACY AND EFFECTIVENESS

Monday, July 15, 2019 4:15 PM – 5:45 PM

O06.1 GENTAMICIN FOR PHARYNGEAL GONORRHEA: A SINGLE-ARM, NON-BLINDED CLINICAL TRIAL

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10.1136/sextrans-2019-sti.134

Background CDC guidelines recommend gentamicin for the treatment of gonorrhea in cephalosporin-allergic patients. The efficacy of gentamicin in the treatment of pharyngeal gonorrhea, which is often undiagnosed, is uncertain.
Methods Between September 2018 – March 2019, we enrolled MSM with NAAT-diagnosed pharyngeal gonorrhea in a single-arm, unblinded clinical trial. Men received a single 360 mg intramuscular (IM) dose of gentamicin at enrollment and underwent test-of-cure (TOC) by culture 4–7 days later. The study measured creatinine at enrollment and TOC, serum gentamicin concentration post-dose to establish peak concentration (Cmax), and standard antimicrobial minimal inhibitory concentrations (MIC). The trial was designed to establish a point estimate for the efficacy of gentamicin for pharyngeal gonorrhea. We planned to enroll 50 evaluable subjects; assuming that gentamicin was 80% efficacious, the trial would establish a 95% confidence interval of 66%-90%. We planned interim analyses at n=10 and n=25.
Results The study was stopped early due to poor efficacy. Of 13 enrolled men, 10 were evaluable, and only two (20%, 95%CI: 2.5% - 55.6%) were cured. Of 2 concomitant rectal infections, both were cured. Efficacy was not associated with gentamicin Cmax ($p=0.809$) or MIC ($p= 0.429$). No participants experienced renal insufficiency; average creatinine percent change was 5% (range: -7%, 21%). Six (46%) subjects experienced headache; all deemed unrelated to treatment. On a scale of 0–10, mean injection pain was 2 (range: 1–7). Among subjects with history of bicillin (n=7) and/or ceftriaxone (n=8) IM injections, 86% believed IM gentamicin to be less painful than bicillin and 75% believed it was more painful than ceftriaxone.
Conclusion 360 mg of gentamicin failed to eradicate *N. gonorrhoeae* from the pharynx. Caution should be used when using the CDC's current alternative therapy (gentamicin 240 mg plus azithromycin 2g) given increases in azithromycin resistance and gentamicin's poor efficacy at the pharynx.
Disclosure No significant relationships.