Results 64 patients participated (median CD4 417/mm³, range 84–1100). 50 of the patients were treated with single dose BPG. Only one patient had ANS (prevalence 1.56% 95 CI 0.04–8.4) with CSF RPR negative, CSF TPPA 1:1280 and lymphocytes 45/mm³. Two patients had a pleocytosis (50 and 22 white cells/mm³ respectively) with negative CSF RPR and CSF TPPA and thus did not meet diagnostic criteria for ANS per protocol.

Discussion/conclusion Our study suggests that single dose BPG is effective treatment for early syphilis in HIV co-infected patients. We will present more data to support this conclusion.

Background/introduction Aerobic vaginitis (AV), a syndrome of abnormal vaginal microflora, was first described in 2002 and is increasingly recognised as a condition distinct from bacterial vaginosis that may require different management.

Aim(s)/objectives To describe the prevalence of moderate-to-severe AV, its management and outcomes in a UK setting.

Methods We included all women presenting to our large integrated sexual health service who met criteria for gynaecological examination and near-patient microscopy. A single biomedical scientist scored the wet mount according to the method of Donders et al. If the score was 5 or above (indicating moderate to severe AV) the requesting clinician was informed. We reviewed case notes to determine treatment choice and outcome.

Results From 1/12/13 to 30/11/14, 1616 wet films were read. Overall, 314 (19.4%) had an abnormal AV score (11 (0.7%) severe AV (score >6), 61 (3.8%) moderate AV (score = 5–6), 253 (15.7%) slight AV (score = 3–4)). Patients with severe AV were significantly older than those with moderate AV (mean age 42.7 vs 32.0 years, p = 0.04), but only 6 (8.3%) patients had atrophic change. Among patients with AV scores of 5 or more, trichomonas was seen in 2 (2.8%) patients, 13 (18.5%) had evidence of yeast infection. First-line treatment included intravaginal clindamycin (49.7%), oral metronidazole (27.3%), antifungals, penicillins, acidification gel and local oestrogen. Resistance to at least one antifungal agent was seen in 26% of patients. Of these, 75% were treated with a second antifungal agent. The overall treatment cure rate was 84.6%.

Discussion/conclusion Patients with moderate-to-severe AV scores are challenging to manage with a high proportion of repeat attendance. Severe AV occurs in an older population.

Clinical Case Studies: 2nd June 2015

CASE SERIES: MANAGING DESQUAMATIVE INFLAMMATORY VAGINITIS IN TRANS-MEN

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Background/introduction Desquamative inflammatory vaginitis (DIV) is an uncommon condition characterised by florid vaginal inflammation causing vaginal discharge, vulval pain and dyspareunia. Microscopy typically shows absent vaginal flora, numerous polymorphs and immature parabasal cells with no mature epithelia. The pathogenesis of DIV is currently unknown but may involve tissue kallikrein-related peptides which are regulated by sex hormones and corticosteroids.

Case 1: 35-year-old trans-man on testosterone for 18-months presenting with yellow vaginal discharge, vestibular pain and dyspareunia. Examination revealed vaginal inflammation and mucopurulent discharge. Microscopy was typical of DIV. He was treated with intravaginal clindamycin reporting a good response.

Case 2: 26-year-old trans-man on testosterone for 7-years presenting with vaginal discharge, dyspareunia and post-coital bleeding. Examination revealed inflamed friable vaginal mucosa. Microscopy findings were typical of DIV and he started treatment with intravaginal clindamycin (partial-response) and switched to intravaginal prednisolone.

Case 3: 20-year-old trans-man with vaginal discharge and post-coital bleeding who started testosterone 6-months earlier. Examination and microscopy findings were typical of DIV. He commenced treatment with intravaginal clindamycin (partial-response) and switched to intravaginal prednisolone.

Case 4: 19-year-old trans-man on testosterone for 9-months presenting with vaginal pain and bleeding. Examination and microscopy were typical of DIV. He started treatment with intravaginal clindamycin (partial-response) and switched to intravaginal prednisolone.

GONOCOCCAL TENOSYNOVITIS IN TWO HIV-INFECTED HETEROSEXUAL MALES: DELAYED DIAGNOSES FOLLOWING NEGATIVE URINE NAAT TESTING

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Background Disproportionately high gonococcal incidence rates amongst men have altered the clinical picture of disseminated gonococcal infection (DGI). The ‘classical’ female patient experiencing a triad of arthritis, tenosynovitis and cutaneous lesions no longer predominates. We present two cases emphasising the need for thorough investigation with evident clinical signs of DGI.

Cases A 48 year old Nigerian heterosexual male presented with a 6 cm inguinal mass and oral hairy leukoplakia. Impression was of lymph node abscess; HIV testing was positive. Urine Nucleic Acid Amplification Testing (NAAT) for chlamydia and gonorrhoea (CT/GC) was negative. Subsequently he developed a swollen tender left wrist. Inguinal abscess aspiration for NAAT testing returned a positive gonococcal result. Treatment was instigated with intravenous ceftriaxone for 4 days, subsequently switching to cefixime for a further week. 3 weeks later his wrist swelling resolved.
A 50 year old HIV-positive British heterosexual male presented after returning from Thailand. He had developed a tender swollen left wrist. Urine NAAT for CT/GC was negative. He reported condomless oral and vaginal sex with multiple Thai females. Gonococcal tenosynovitis was suspected and extragenital NAATs and cultures for CT/GC were taken; NAAT for pharyngeal gonorrhoea was positive. Single dose ceftriaxone and azithromycin was prescribed, followed by cefixime for 2 weeks. Two weeks later his symptoms cleared.

**Conclusion** Reflecting on these cases a DGI diagnosis was attained following careful consideration of possible differentials and persistence in identifying *Neisseria gonorrhoeae*. Both diagnoses would have been missed if following current testing guidance which recommends penile-only sampling of heterosexual men.

**Background** A 38 year old man presented for HIV testing following his male partner’s diagnosis. Examination revealed systolic and decrescendo diastolic heart murmurs, palpable thrill, bounding pulses, and positive Corrigan’s sign. He had not tested previously for HIV or syphilis and had been in a monogamous relationship for 8 years. We describe this man who was asymptomatic – from both HIV and aortic valve disease – with incidental diagnosis of severe syphilitic aortitis following partner notification for HIV.

**Results** HIV antibody test was positive with baseline viral load 239505 copies/ml and CD4 count 103 cell/µL (8%). Syphilis serology was positive with rapid plasma reagin (RPR) 1:4. CXR was unremarkable. ECG was consistent with left ventricular hypertrophy with strain. Echo revealed severe mixed aortic valve disease, left ventricular hypertrophy, good LV systolic function and normal aortic arch appearance. He commenced prednisolone 60 mg OD for 5d, 72 hr before starting three weekly doses of 2.4 MU benzathine penicillin. He was admitted for 48 hr for cardiac monitoring at the start of treatment which proceeded without a complication. Multidisciplinary involvement with GU physicians, cardiologists and cardiothoracic surgeons was instigated from the start with aortic valve ± root replacement planned imminently.

**Discussion** Resurgence of syphilis in the UK was reported in the late 1990s with an ongoing epidemic since, mainly involving MSM. Cardiovascular syphilis typically occurs 15–30 years following primary infection with *Treponema pallidum*, with complications in 10% of cases. Could this man be amongst the first cases to develop tertiary syphilis in this latest epidemic?

**Background** Vulvovaginal candidiasis (VVC) is a common condition caused by *Candida albicans* in 80–92%. *Candida robusta* is rarely identified in humans and has only been reported as a cause of VVC in pregnant women. We present a case of chronic *Candida robusta* VVC.

**Case** A 25 year-old, on Cervazette, presented to her GP with discharge and vulval itching; treatment with clotrimazole was effective but symptoms recurred. In clinic, one month later, a clinical and microscopic diagnosis of VVC was made, she was treated with fluconazole plus econazole pessary and cream. HIV, syphilis, gonorrhoea and chlamydia were negative.

**Conclusions** Despite initial improvement she represented with recurrent symptoms, microscopy and culture again confirmed Candida species. Following a fourth presentation oral fluconazole 150 mg every 72 h x 3 followed by a weekly dose for three months was commenced. She was asymptomatic during this time but relapsed on discontinuation. Microscopy again confirmed spores and on speciation Candida robusta sensitive to fluconazole was isolated. A second 3-month fluconazole course was given. She had now developed provoked vulvodynia. Low-grade symptoms persisted and Candida robusta was again cultured, now resistant to fluconazole. A one-week course of oral voriconazole was given. Follow-up microscopy was negative but her vulvodynia had worsened. Treatment with amitriptyline was commenced and on review two months later culture remained negative and her vulvodynia had improved.

**Discussion** We report a case of chronic Candida robusta VVC in a non-pregnant immunocompetent woman, which acquired fluconazole resistance and precipitated vulvodynia. Speciation and sensitivity testing are important in women with recurrent symptoms.