

**Case** We present the case of a 41-year-old MSM who was admitted with fever, confusion and agitation following unprotected anal sex 6 weeks earlier with a man of unknown HIV status. He required intubation due to the level of agitation. Admission CT and MRI brain were unremarkable. The first test HIV test was weakly antibody positive; a repeat 6 days later showed a stronger antibody response consistent with HIV seroconversion. His baseline CD4 count was 138 (18%); HIV viral load was 929 000 copies/ml. CSF analysis showed 4 white blood cells, elevated protein at 1.2 g/dl, and a normal CSF to plasma glucose ratio. There was insufficient CSF sample for HIV viral load testing. A diagnosis of encephalitis secondary to HIV seroconversion was made and antiretroviral therapy (ART) with five drugs was started on day 3 of admission. The patient remained agitated for several days. By day 21 the seroconversion symptoms had fully resolved, and by day 28 the plasma HIV viral load was undetectable. Viral genotyping showed the K103N mutation only. The patient remains on ART; now simplified to Kivexa, Darunavir and Ritonavir. This is to continue for 48 weeks at which point a decision will be made to either stop treatment or to continue lifelong ART.

**Conclusion** Transmitted drug resistant HIV can cause severe seroconversion illness and high levels of viraemia despite lower viral fitness. To our knowledge, this is the first report of viral encephalitis at HIV seroconversion caused by drug resistant HIV. The role of entry inhibitors and integrase inhibitors for the treatment of severe seroconversion symptoms to prevent viral entry into cells and aid rapid decline in viraemia are currently under evaluation.

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#### CASE REPORT: RARE CAUSE OF ADULT ONSET SEIZURES IDENTIFIED IN AN HIV POSITIVE ADULT

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<sup>1</sup>E Pease, \* <sup>1</sup>S Dawson, <sup>2</sup>G Quaghebeur, <sup>1</sup>J Ashby. <sup>1</sup>The Garden Clinic; <sup>2</sup>John Radcliffe Hospital, Oxford, UK

**Case Report** A 63-year-old male with a long history of poor social interactions presented acutely confused. There was no alcohol or recreational drug use. Baseline bloods and routine microbiology were normal. CT and MRI of the brain found no causative pathology. Lumbar puncture (LP) only revealed a raised protein (1.47). An HIV test was positive; CD4 170 cells/ $\mu$ l, viral load (VL) 238 281 copies/ml. The patient refused antiretroviral (ARV) treatment, depressive episode was diagnosed and mirtazapine commenced. Three days later he went into status epilepticus. A repeat MRI brain was unchanged and LP showed: protein 0.84; glucose 4; WBC 5/cmm; India ink, CRAG, virology, acid fast bacilli and cytology were negative. Seizures were unremitting and nevirapine and zidovudine were commenced and mirtazapine withdrawn: seizure activity ceased. Gradually he improved and his CD4 rose to 280 cells/ $\mu$ l, HIV VL <50. Despite treatment, the patient re-presented with seizures on several occasions. No trigger was identified, and reported ARV adherence good. Repeat investigations revealed no new abnormality. In the absence of an adequate explanation for seizures, neuroimaging was reviewed by a specialist neuroradiologist. Nodules within the frontal horns of the lateral ventricles were identified, indicating a diagnosis of subependymal nodular heterotopia (SNH).

**Discussion** This is the first report of SNH in an HIV positive adult. SNH are congenital, occurring during foetal neuronal proliferation and have no known association with HIV. Patients usually present in their 2nd decade with seizures and exhibit cognitive difficulties. SNH presenting in the 7th decade of life with status epilepticus is rare and we postulate that the cerebral atrophy associated with untreated HIV infection may have precipitated seizures in a predisposed individual. This case described demonstrates the importance including non-HIV related causes, when considering the aetiology of seizures in patients with HIV.

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#### THE TANGLED MESH OF LYMPHOGRANULOMA VENEREUM

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I Okpaluba, \* R J C Dunham, A Evans. Leeds Teaching Hospitals NHS Trust, Leeds, UK

**Introduction** The HPA report an epidemic of lymphogranuloma venereum (LGV) among men who have sex with men in the UK since 2003. Untreated LGV may lead to chronic or irreversible complications with disabling anatomic defects. Reports suggest surgical intervention is rarely required.

**Case** 45-year-old man who has sex with men, diagnosed HIV positive in 1995, not on antiretroviral therapy due to patient reluctance, following intolerance to several previous regimens. He had spent the summer in Egypt and on return to the UK had a brief spell as an inpatient with campylobacter and norovirus diarrhoea. He presented to the GUM clinic reporting multiple high risk partners and was treated as a gonorrhoea, chlamydia and syphilis contact; STI screen negative. He re-presented a month later with severe proctitis; pain and blood per rectum and frequent small volume stool. Rectal chlamydia was positive and subsequently LGV serovars confirmed. He tested positive for hepatitis C, retrospective sampling of stored blood samples suggested that this had been recently acquired. Hepatitis C treatment was unsuccessful due to lack of virological response and he suffered acute psychosis likely secondary to pegylated interferon. Despite 3 weeks of doxycycline and negative chlamydia retesting he persisted with severe proctalgia and constipation and was referred to gastroenterology. Flexible sigmoidoscopy showed a single ulcer in the upper third of the rectum with the remainder of the colon looking normal. He received empirical retreatment of LGV, rectal predfoam and laxatives. Symptoms worsened and a defecating proctogram (video of proctogram available) identified marked anorectal intussusception. This was surgically managed with a mesh support inserted laparoscopically.

**Conclusion** Despite early recognition, treatment and apparent clearance of LGV infection complications can occur. This is the first report of complicating anorectal intussusception requiring surgical intervention with a mesh support.

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#### THE GREAT PRETENDER STRIKES AGAIN

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<sup>1</sup>T Moby, \* <sup>2</sup>A Mwirigi, <sup>1</sup>J White. <sup>1</sup>St Thomas' Hospital; <sup>2</sup>University Hospital Lewisham, London, UK

**Background** An epidemic of syphilis persists in UK men who have sex with men (MSM), often with unusual manifestations. We report a case of syphilis mimicking a lymphoproliferative disorder.

**Case** A 29-year-old previously well MSM was admitted with 3 weeks of generalised painless lymphadenopathy. He reported malaise, night sweats and joint pain while travelling recently in the Middle East. He was afebrile and routine bloods were normal apart from mildly raised liver enzymes. Paul Bunnell and HIV antibody tests were negative. His GP screened for STIs but did not include syphilis serology despite the patient describing penile lesions. Clinicians felt that the presentation was highly suggestive of lymphoma. A CT scan showed multiple enlarged lymph nodes in the neck and small bowel mesentery. An open cervical lymph node biopsy was performed. Histopathology showed suppurating granuloma in a reactive lymph node with no evidence of lymphoma. Stains for HIV p24, acid-fast bacilli and fungi were negative. The suggested differential included lymphogranuloma venereum (LGV), cat scratch disease and melioidosis. At GU medicine review he reported sex with multiple partners in the preceding 6 months. He had a blotchy maculopapular rash on his penis and scrotum, though this was treated as "fungal" by junior staff. Molecular tests for