nor risk factors for HEV. Although HEV serology was initially equivocal, IgG and IgM later became positive with detectable PCR. His ALT normalised and HEV-PCR became undetectable four weeks later.

**Discussion/conclusion** HEV appears to be a self-limiting-asymptomatic illness in HIV+ MSM with good CD4 counts. HEV may be sexually transmitted in populations with increasing STI rates. HEV should be considered a potential cause of elevated liver enzymes in HIV+ patients.

**P35 ARE TESTICULAR MIXED GERM CELL TUMOURS ASSOCIATED WITH HEPATITIS C(HCV) IN HIV INFECTED MEN WHO HAVE SEX WITH MEN?**

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**Background/introduction** HCV infection and testicular germ-cell tumours are indicator diseases for HIV-testing in BASHH-guidelines. There is little data on the association of testicular tumours in MSM with HIV.

**Aims/objectives** We describe 2 MSM with treated HIV-Hepatitis C co-infection who were both subsequently diagnosed with mixed-germ-cell testicular tumours.

**Case details** Patient-1 is a 51-year-old MSM, diagnosed with HIV in 2004 on Atripla since 2010. In May 2012, routine ALT = 186 and positive HCV-RNA (genotype 1). This was treated with 48-weeks of pegylated-interferon/ribavirin. He had a sustained-viral-response (SVR). Two years later, he presented to the STI-clinic with a four month history of testicular swelling. Ultrasound showed this to be likely malignant infiltration, AFP = 2484, LDH = 426, HCG = 5.9. After orchidectomy, histology demonstrated mixed germ cell tumour. He is in clinical/radiological remission.

**Conclusion** Two years later, he presented to the STI-clinic with a four month history of testicular swelling. Ultrasound showed this to be likely malignant infiltration, AFP = 2484, LDH = 426, HCG = 5.9. After orchidectomy, histology demonstrated mixed germ cell tumour. He is in clinical/radiological remission. Patient-2 is a 41-year-old MSM diagnosed with HIV in 2007 he received IL-2 in a clinical trial. In both 2008 and 2012 routine ALT = 918,505 respectively and HCV-RNA was positive (genotype 2/3)(genotype 1). HCV was treated with pegylated-interferon/ribavirin both times with SVR. Anti-retrovirals (Atripla) were started in 2012. That year, he presented with an E-Coli-UTI and testicular swelling. Ultrasound/ orchidectomy found a mixed germ cell tumour. He had a sustained-viral-response (SVR). Two years later, he presented to the STI-clinic with a four month history of testicular swelling. Ultrasound showed this to be likely malignant infiltration, AFP = 2484, LDH = 426, HCG = 5.9. After orchidectomy, histology demonstrated mixed germ cell tumour. He is in clinical/radiological remission.

**Discussion/conclusion** HIV infection and hepatitis C treatment are immunosuppressive and are potential causative factors in these HIV-MSM testicular germ-cell tumours. Early investigation of testicular swellings in men with HIV-Hepatitis C is important.

**P37 SEXUALLY ACQUIRED SALMONELLA TYPHI URINARY TRACT INFECTION**

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**Case report** A 22 year old MSM was diagnosed HIV positive with a CD4 count was 475 cells/mm3 (35%). He suffered urinary symptoms and Salmonella typhi was isolated from urine culture. He recalled a self-limiting afebrile diarrhoeal illness 2 weeks earlier. Stool and blood cultures were negative. He completed a one-week course of ciprofloxacin with subsequently negative cultures. He had no past medical history or significant travel history. He reported unprotected anal intercourse one month before HIV diagnosis, and protected anal intercourse with several partners since diagnosis, but no other infections have been reported locally. All named contacts have declined testing.

**Discussion** The most common manifestation of S.typhi infection is typhoid fever. Most cases in the developed world have been acquired through faeco-oral transmission in endemic areas. Haematogenous dissemination can be widespread and more severe among the immunocompromised. Death ensues in up to 32%. Infection of the genitourinary system is rare. Cases reported have a background of urinary tract abnormalities, invariably with blood and/or stool culture positivity. There are no cases in the literature of sexually acquired S.typhi UTI. Infections were acquired through oro-anal contact and pathogen ingestion. None had UTI. Our patient had repeatedly negative blood and stool cultures, reducing the likelihood that this was a disseminated infection leading to UTI, and raising the possibility that the route of infection was though insertive anal intercourse with direct urethral inoculation with S.typhi. Unfortunately partner notification has not identified an infected sexual contact to add further weight to this theory.