

results were maintained. Among the HIV disease biomarkers including viral load blips over the study period, only a lower nadir was associated with decreasing Creatine (Spearman Rho $r = -0.49$, $p < 0.03$).

Conclusions Chronic HIV infection is associated with subclinical cellular energy abnormalities (Creatine) linked to past levels of immunosuppression and acceleration of neuronal integrity (N-Acetyl Aspartate). This preliminary study further reinforces the need for longer-term follow-up in chronic HIV-infected ageing persons to determine the prognostic value of these findings.

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022 - Ending hepatitis C in populations

022.1 AUSTRALIAN HIV/HEPATITIS C CO-INFECTED PATIENTS FALL BEHIND HIV MONO-INFECTED PATIENTS IN MOVE TOWARDS EARLY HIV TREATMENT INITIATION

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Introduction One in eight people living with HIV (PLHIV) in Australia are estimated to be co-infected with Hepatitis C (HCV). In the light of clinical guideline changes with regard to antiretroviral therapy (ART) initiation, we examine the impact of co-infection status on uptake of treatment, and highlight population-specific differences relative to HIV mono-infected patients.

Methods Ipsos Healthcare's HIV Therapy Monitor is a patient chart audit study, which monitors trends in the treatment of PLHIV in Australia. Demographic and treatment data are collected bi-annually from a panel of 25+ HIV-treating clinicians. The data in this report is based on a sample of 4331 patient records collected between 2008- 2014, of which 412 were co-infected with HCV.

Results While the proportion of HIV mono-infected patients receiving ART has steadily increased from 67% in 2008 to 84% in 2014 ($p < 0.0001$), the opposite trend is observed in the HIV/HCV co-infected population. The rate of treatment in the co-infected cohort has dropped from 84% in 2010/2011 to 66% in 2014 ($p = 0.003$), with co-infected patients experiencing an average delay of 42 months between HIV diagnosis and initiation of ART, compared with 25 months for mono-infected patients ($p = 0.013$). Patient's lack of support network was most frequently cited by clinicians as the reason for delaying treatment for co-infected patients, followed by patient choice and expected non-compliance.

Conclusion Increasing evidence is now available to support early initiation of ART, both in terms of clinical benefits as well as in preventing disease transmission. However, despite encouraging results among HIV mono-infected patients, outcomes for the HIV/HCV co-infected population reveal a growing disparity between these groups in Australia. The increasing delay to treatment supports the need to consider this patient group a priority population, and indicates that further action is required to address the complications involved in treating these patients.

Disclosure of interest statement There are no conflicts of interest to declare.

022.2 DETECTION OF HEPATITIS C VIRUS (HCV) IN SEMEN FROM HIV-INFECTED MEN WHO HAVE SEX WITH MEN (MSM) DURING ACUTE HCV INFECTION

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Introduction The mechanism(s) and bodily fluid(s) involved in the recently identified epidemic of sexually transmitted HCV in HIV-infected MSM are unclear. HCV is present only intermittently and at low levels in semen from men with chronic HCV-infection, however little is known of the dynamics of seminal HCV during acute HCV-infection.

Methods HIV-infected MSM with acute and chronic HCV-infection were prospectively enrolled into an IRB-approved study. Three paired semen and blood specimens were collected at 2-week intervals. HCV viral load (VL) was quantified using an automated RT-PCR assay platform (Abbott).

Results Paired semen and blood specimens were obtained from 33 HIV-infected MSM (21 with acute-HCV and 12 with chronic-HCV). Sixteen (27%) of 59 semen specimens had detectable HCV VL, with 11 (33%) men having at least one positive specimen. Semen specimens with detectable HCV had a significantly higher median blood HCV VL ($P = 0.002$). There were no differences between men with acute or chronic HCV in either the proportion of semen specimens positive for HCV (8/38 [21%] and 8/21 [38%], respectively; $P = 0.159$), or in the median seminal HCV VL (1.32 log IU/ml and 1.77 log IU/ml, respectively; $P = 0.163$).

Conclusion This study, although identifying no differences in the magnitude or proportion of seminal HCV during acute HCV-infection, provides valuable insights into the dynamics of seminal HCV during this period. It is unknown whether the levels of seminal HCV identified in this study are sufficient for the sexual transmission of HCV in HIV-infected MSM. However, it is plausible that HCV in semen deposited in the rectum after the friction of receptive anal intercourse, could enter the blood stream and infect the liver. Future research should focus on establishing the infectivity of seminal HCV, and the analysis of seminal HCV levels during the 'ramp-up' period of early acute HCV-infection, where blood HCV levels are highest.

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