358. HIV Infection and the Risk of Hepatocellular Carcinoma in Patients with Hepatitis B Virus (HBV) Co-infection: a Propensity Score-matched Cohort Study
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**Session:** 45. HIV Complications: Hepatitis Co-Infections
**Thursday, October 3, 2019: 12:15 PM**

**Background.** There is a paucity of data to show whether HIV infection would affect the risk of hepatocellular carcinoma (HCC) in patients with chronic hepatitis B virus (HBV) infection.

**Methods.** A territory-wide cohort study was performed to determine the risk of HCC in patients with HBV with and without HIV co-infection. All patients with HBV/ HIV co-infection and HBV mono-infection treated with antiviral therapy in public hospitals in Hong Kong from 2000 to 2017 were identified from an electronic database. Patients with hepatitis C virus (HCV) infection, HCC diagnosed within six months, or follow-up less than 6 months were excluded. The primary outcome was HCC. A propensity score (PS) for each patient was defined as the conditional probability of having HIV infection given the baseline characteristics (including age, sex, cirrhosis, bilirubin, alanine transaminase/ALT, platelet, albumin, and prothrombin time). HBV/HIV-co-infected and HBV-monoinfected patients were matched in a 1:5 ratio by PS matching. Weighted Fine-Gray subdistribution hazards model was estimated, where the variables included were HIV status and ALT as the other important co-variates were well matched.

**Results.** A total of 822 HBV/HIV-co-infected and 53,974 HBV-monoinfected patients were identified, and 692 and 38,102 were included for PS matching (Figure 1). Six hundred and three HBV/HIV-coinfected and 2,380 HBV-monoinfected patients were included in the final analysis. Among this cohort, 85% were male, mean (± standard deviation) age was 42 ± 12 years, and 4.5% had cirrhosis at baseline. At a median follow-up of 5.8 (interquartile range 2.6–9.6) years, 7 (1.2%) and 75 (3.2%) HBV/HIV-coinfected and HBV-monoinfected patients developed HCC, respectively. Weighted Fine-Gray model showed that HIV infection was associated with a lower risk of HCC (subdistribution hazard ratio 0.39, 95% confidence interval 0.16–0.94, P = 0.036) (Figure 2).

**Conclusion.** HIV/HBV co-infected patients had lower risk of HCC compared with antiviral therapy-treated HBV-monoinfected patients. This observation can be explained by a lower threshold, in terms of severity of liver disease, to start antiviral treatment in HBV/HIV-coinfected compared with HBV-monoinfected patients.
previously shown to influence the rate of fibrosis progression. Hepatic fibrosis change was determined using the serum-derived Enhanced Liver Fibrosis (ELF) Index. Four putative genes with polymorphisms that have been previously associated with the development or progression of hepatic fibrosis were evaluated using Taqman SNP genotyping assays. Cytokine assays were performed using LumineX chipsets. Samples were analyzed using Statistic 10.0 using ANOVA and least square regression models.

**Results.** 58 unique subjects were evaluated. The mean age was 38 years, and all were male. 74% were HIV infected and 97% were HCV infected (76.8% coinfection). Controlling for the effect of CCR5, only the TR2L A -> G polymorphism was predictive of change in the ELF Index. There was no statistically significant predictive difference between genotypes in the other three polymorphisms. Subjects with the TR2L A allele (n = 47) had an average increase in ELF of 0.79 units, while the G allele (n = 11) had an increase in ELF of 2.1 units (P = 0.008). A regression model identified TR2L as a key factor in ELF change, as well as HCV/HIV coinfection. Interferon alfa-2 levels were highly associated (increased, P = 0.0007) with the TR2L A -> G polymorphism, while RANTES levels were inversely associated (decreased, P = 0.0443) with it.

**Conclusion.** Of the gene polymorphisms investigated, only TR2L (rs179009) is an independent predictor of development of hepatic fibrosis in HCV/HIV coinfected subjects. The mechanism may involve modulation of inflammatory response pathways.

**Disclosures.** All authors: No reported disclosures.

### 360. Advanced Liver Disease in HIV/Hepatitis B Coinfected Patients: Associated with Race, Age, and Comorbidities

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**Session:** 45. HIV Complications: Hepatitis Co-Infections

**Background.** Hepatitis B virus (HBV) coinfection is common in people with HIV. Compared with HBV mono-infected individuals, those that are HIV/HBV coinfected show evidence of more rapid progression to advanced liver disease (ALD) and increased mortality rate. In this study, we identified characteristics in an HIV/HBV cohort associated with ALD.

**Methods.** We retrospectively examined an HIV/HBV coinfected cohort to determine the prevalence of ALD and its correlation with selected variables. Data were drawn from HIV and HBsAg+ patients at three HIV clinics in Houston, Dallas, and San Antonio, Texas. Those without chronic HBV were excluded. ALD was defined as cirrhosis, decompensation, and/or hepatocellular carcinoma, as determined by imaging, and associated with increased risk for ALD.

**Results.** Within those with HIV/HBV coinfection (n = 501), 89 (18%) met the criteria for ALD (92% male, 47% Black, 33% White, 16% Hispanic, 5% > 40 years old). Amongst these (n = 89), significant differences were observed with race (P = 0.039), age (P = 0.001), patients identified as MSM/Bisexuals (P = 0.047), diabetes mellitus (DM) (P = 0.01) and hepatitis C virus (HCV) coinfection (P = 0.001). Compared with Whites, Blacks are less likely to have ALD (95% CI 0.27, 0.79, P = 0.004), and those age 40-49 (95% CI 1.28, 10.92, P = 0.016) and ≥50 (95% CI 1.63, 15.54, P = 0.005) were more likely. The multivariate logistic regression analysis showed patients that are White race, age ≥50, have DM, and those with HCV coinfection had increased risk for ALD (Table 1). No differences were seen with gender, insurance, alcohol use, HBsAg loss, HepB eAg status or baseline CD4 count, HBV DNA, and HIV RNA viral load. Bivariate analysis suggested the following risk factors: Whites, elder age (>50), and comorbidities of DM and HCV. These should be taken into consideration when approaching the development and treatment of ALD in HIV/HBV patients.

**Disclosures.** All authors: No reported disclosures.

| Variables | Adjusted Odds Ratio | 95% CI | P value |
|-----------|---------------------|--------|---------|
| Race/Ethnicity |                      |        |         |
| White    | 0.51                | 0.29, 0.99 | 0.02 |
| Hispanic | 0.73                | 0.34, 1.55 | 0.41 |
| Other    | 1.15                | 0.33, 3.89 | 0.82 |
| Age group ≥ 50 years | 1.46   | 0.46, 4.60 | 0.52 |
| 30-39 years | 2.87                | 0.86, 8.63 | 0.03 |
| 40-49 years | 3.79                | 1.16, 12.38 | 0.04 |
| Diabetes | 1.98                | 0.38, 10.23 | 0.07 |

**Disclosures.** All authors: No reported disclosures.

### 361. Residual Lamivudine-Resistant Hepatitis B Virus Detected on Next-Generation Sequencing of Treatment-Experienced HIV Patients Failing Antiretrovirals

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**Session:** 45. HIV Complications: Hepatitis Co-Infections

**Background.** Hepatitis B is highly prevalent in the Philippines, with 17% of the population infected. With the fastest-growing HIV epidemic in the Asia-Pacific, 12% of HIV patients are HBsAg reactive. With the use of lamivudine and tenofovir-based antiretrovirals (ARVs), hepatitis B virus (HBV) treatment in co-infected HIV patients is not usually an issue. However, there is a potential to develop HBV resistance when patients are switched off tenofovir when antiretroviral resistance develops. With high rates of acquired K65R tenofovir resistance, the potential for re-emerging HBV resistance is present. We report two HIV patients with residual whole-genome HBV with lamivudine and tenofovir resistance mutations.

**Methods.** As part of a surveillance study on acquired drug resistance in the Philippines, samples with an HIV viral load >1,000 copies underwent Sanger sequencing of RT and PR for genotyping and HIV drug-resistance testing. Near-whole-genome next-generation sequencing (NGS) for HIV using Illumina HiSeq was also performed on these samples.

**Results.** Two patients had coincidental whole-genome amplification of HBV on NGS (Table 1). HBV serology for both showed reactive anti-HBsAg and non-reactive HBsAg and Anti-HBc. The two HBV samples were genotype A and were resistant to lamivudine and tenofovir, with intermediate resistance to entecavir.

**Conclusion.** Residual HBV may be present in patients on ARVs. Antibody responses for HBV serology may not be very reliable in highly immunosuppressed patients. The potential of lamivudine-resistant HBV to emerge when HIV patients are shifted off tenofovir due to resistance in patients should be considered when deciding on second-line ARVs.

**Disclosures.** All authors: No reported disclosures.

| Variable | Baseline CD4 count (cells/mm³) | Nadir CD4 count (cells/mm³) | Baseline HIV viral load (copies/mL) | HIV genotype | HIV mutations | Initial ART Regimen | Revised ART Regimen | HBV viral load (copies/mL, limit of detection is 179) | HBV Genotype |
|----------|--------------------------------|-----------------------------|-----------------------------------|--------------|---------------|--------------------|-------------------|-----------------------------------------------|--------------|
| Case 1   | 229                            | 1,001,000                   | 7                                 | PR: CPRI/AL   | NRTs: K65R     | tenofovir/lamivudine/efavirenz |        | undetectable          | A             |
| Case 2   | 45                             | 97,000                      | 45                                | PR: CPRI/AL   | NRTs: K65R     | tenofovir/lamivudine/efavirenz |        | undetectable          | A             |

**Disclosures.** All authors: No reported disclosures.

### 362. Hepatitis C Virus (HCV) Co-Infection in Women Living with Human Immunodeficiency Virus (HIV) in Northwest Louisiana

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**Session:** 45. HIV Complications: Hepatitis Co-Infections

**Background.** HIV and HCV infection are emerging global public health problems. People living with untreated HCV infection have higher HCV viral loads and more rapid HCV disease progression with twice the rates of perinatal HCV transmission. Data are lacking in HCV coinfected women living with HIV. Our study reviewed underrepresented minority group of women living with HIV/HCV in Northwestern Louisiana to better understand epidemiology, risk factors and access to care among our cohort.

**Methods.** Women with HIV/HCV coinfection aged 18-70 years who presented to an academic medical center between November 2011 and November 2018 were included for analysis. A retrospective chart review was conducted. Data were collected and analyzed on demographics (age, race), risk factors (sexual history, drug use), HIV