355. Barriers for Hepatitis C Elimination in HIV/HCV Coinfected Patients
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Background. Approximately 30% of people living with HIV are co-infected with Hepatitis C virus (HCV). HIV/HCV coinfection fortunate partners have faster progression to liver fibrosis, cirrhosis, and increased mortality, compared with monoinfected patients. Therefore, treatment in this population is a priority. The objective of this study was to develop an active program to reach HIV/HCV co-infected patients, with the goal to eliminate Hepatitis C in our local HIV clinic.

Methods. Beginning in December 2016, our clinic received State funds to support open access to treat HIV/HCV patients with direct-acting antivirals (DAA). From December 2016 to May 2018, the process was based on primarily on physician referral to treat HIV/HCV patients at our clinic, without an active intervention, and 50 patients were treated. Our active intervention during the second part was based on the identification of all untreated HIV/HCV patients and contacting them directly, to link them to care.

Results. A total of 462 HIV/HCV co-infected patients were identified who qualified for the state-sponsored treatment program. From June 1, 2018 to July 31, 2018, only 7 patients were linked to care and started on DAA. The four main identified reasons for not getting DAA therapy were: no show up to the clinic appointments, poor adherence to their HIV antiretroviral treatment, use of drugs and not able to reach (figure). Although drug use was listed as one of the main reasons for not receiving DAA therapy, it was not the defining reason for most patients. A majority of the patients had more than one obstacle preventing them from coming in to be treated.

Conclusion. Wide availability of DAA and open access to treatment is not enough to eliminate HIV/HCV co-infection. Innovative outreach processes with the active participation of key stakeholders are needed in order to eliminate this viral infection.

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356. HIV-HCV Coinfection: An Investigation of CD4 T-Cell Reconstitution after HCV Direct-Acting Antiviral Treatment
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Background. Both human immunodeficiency virus (HIV) and Hepatitis C virus (HCV) can adversely affect CD4 cell count. Among patients with HIV-HCV coinfection, treatment with HCV direct-acting antivirals (DAAs) can provide the opportunity to restore/reconstitute CD4 count. The primary objective of this study was to determine whether DAA treatment results in improved CD4 cell counts in HIV-HCV coinfected patients.

Methods. A retrospective multicenter cohort study was performed among 4 sites between 1/1/2013–4/12/2018. Patients were included if they were age 218 years, infected with both HIV and HCV, and received all-oral DAA therapy. Trained reviewers extracted demographics, comorbidities, receipt of antiretroviral therapy (ART), DAA treatment regimen/duration and HIV/HCV-related lab values, which included CD4, HIV RNA, and HCV RNA. Labs were restricted to the closest values before/after DAA treatment. The primary endpoint was changed in pre-DAA/post-DAA CD4 count. Descriptive statistic and Wilcoxon Signed Rank were used.

Results. There were 88 patients included. Most (78.4%) identified as male. Mean ± standard deviation (SD) age was 57.1 ± 9.6 years. The proportion of patients with undetectable pretreatment HIV RNA was 78.4%. Among the 97.7% of patients on ART, regimens included the following classes of ART: integrase strand transfer inhibitor (75.6%), non-nucleoside reverse transcriptase inhibitors (23.3%) and protease inhibitors (19.8%). Of the 87 patients who completed DAA therapy and had post-DAA labs drawn, sustained virologic response (SVR) was achieved in 96.6%. The median (interquartile range, IQR) CD4 counts before/after DAA treatment did not significantly differ (515 [349–704] vs. 552 [374–693], P = 0.80). In the subset of pre-DAA CD4 counts < 350 cells/mm³ (n = 23), CD4 count significantly improved before/after DAA treatment [235 [202–311] vs. 309 (189–392), P = 0.01].

Conclusion. The use of DAA therapy in HIV-HCV co-infected patients resulted in a significant increase in CD4 count in patients with pre-DAA CD4 < 350 cells/mm³. This may represent a high priority population for DAA treatment.

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357. Hepatitis C (HCV) Testing and Diagnosis and Their Relationship to Sexually Transmitted Infection (STI) Screening and New Infections in an HIV+ Men Who Have Sex with Men (MSM) Outpatient Cohort
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Background. Assess HCV prevalence, screening, new infections and reinfections and their relationship to other STI screening and diagnosis in HIV+ MSM at an urban center.

Methods. Retrospective review of HCV and STI testing and diagnosis among HIV+ MSM with ≥ 1 visit January 1, 2016–October 31, 2018 at Montefiore’s Center for Positive Living and Medical Group outpatient sites. Demographics from local databases, clinical data from EPIC and chart review on select cases.

Results. 876 HIV+ MSM, median age 42, 80% virally suppressed. 850 (98.2%) had known HCV status. 36/850 (4.2%) HCV Antibody (Ab)+ at any point: 23 (2.7%) at baseline (6 dual MSM/EDU); 13 (1.5%) newly Ab+ (0 dual risk); 43/6 (11.1%) HCV RNA+: 1 of baseline Ab+, 3 newly Ab+. Among new Ab+, 7 asymptomatic, 6 symptomatic, most commonly high liver tests, 3/13 (15.4%) were persistently viremic requiring therapy: 614/872 (74.2%) HCV Ab– were retested 2, 260 (34.4%) >1x average retesting interval 13 months. Among 36 HCV Ab–, 0 had reinfection. Testing and new STIs by HCV status is in Table 1. 2/13 (15.4%) with new HCV were not tested for gonorrhea or chlamydia (G/C) at any site. Acute syphilis was more common in new HCV+ than HCV– (P = 0.02). HCV rescreening was higher in those tested for extragenital (EG) G/C vs. those not tested (Table 2), but up to 18.8% were not HCV retested despite EG testing done. 304/876 (34.7%) were tested 7 times vs. 35 years of age. Testing and positive results for all 4 STIs were greater in those ≥ 35 (Table 3). Non-Hispanic (NH)-Black was the largest race/ethnicity and had the highest rate of new STIs except pharyngeal chlamydia, rectal gonorrhea and acute syphilis (Table 4).

Conclusion. We found significant risk of HCV among HIV+ MSM in our cohort, with a prevalence of 2.7% and a 34-month incidence of 1.5%, with no reinfections. HCV+HSV Ab– MSM were frequently retested for HCV but missed opportunities among sexually active individuals lead to delayed diagnoses of acute infection. Unexplained elevation of liver tests in sexually active HIV+ MSM should prompt immediate HCV testing, and more HCV Ab testing is indicated as part of STI screening in this group. Awareness should be raised about risk of acute HCV with new syphilis, and there is room to improve EG G/C testing.
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358. HIV Infection and the Risk of Hepatocellular Carcinoma in Patients with Hepatitis B Virus (HBV) Co-infection: a Propensity Score-matched Cohort Study Grace Lui1, MBChB; Terry Yip, MSc1; Becky Yuen, MSc1; Alex Sze, MBChB2; Yee-tak Hui, MBBS2; Yee-Kit Tse, MSc1; Vincent Wong, MBChB1 and Grace Wong, MBChB1; 1The Chinese University of Hong Kong, Hong Kong, 2Queen Elizabeth Hospital, Hong Kong

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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 and with 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 six 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-coinfected 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-infected 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.

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359. TLR7 Gene Polymorphisms Influence Development of Hepatic Fibrosis in HCV/HIV Coinfection Kenneth Sherman, MD, PhD1; Heidi L. Meeds, BS2; Enas A. Abdel-hameed, MD, PhD; Susan D. Rouster, BS3; and M Tarek M. Shata, MD, PhD1; 1The University of Cincinnati College of Medicine, Cincinnati, Ohio; 2University of Cincinnati, Cincinnati, Ohio

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Methods. Longitudinal samples were obtained from subjects enrolled in the NCI Multicenter Hemophilia Cohort Study. Within the cohort, a subset of subjects were included based upon presence or absence of the CCR5 delta-32 mutation which was...