	Chronic HCV+ (n=23)	Acute HCV+ (n=13)	HCV- (n=814)
Urine G/C Tested	18/23 (78.3%; 56.3%-92.5%)	11/13 (84.6%; 54.6%-98.1%)	745/814 (91.5%; 89.4%-93.3%)
U-G+	1/18 (5.6%; 0.1%-27.3%)	1/11 (9.1%; 0.2%-41.3%)	46/745 (6.2%; 4.6%-8.2%)
U-C+	2/18 (11.1%; 1.4%-34.7%)	0/11 (0%)	36/745 (4.8%; 3.4%-6.6%)
Pharyngeal G/C Tested	14/23 (60.9%; 38.5%-80.3%)	10/13 (76.9%; 46.2%-95.0%)	500/814 (61.4%; 57.9%-64.8%)
P-G+	6/14 (42.9%; 17.7%-71.1%)	1/10 (10.0%; 0.25%-44.5%)	70/500 (14.0%; 11.1%-17.35%)
P-C+	2/14 (14.3%; 1.8%-42.8%)	0/10 (0%)	24/500 (4.8%; 3.1%-7.1%)
Rectal G/C Tested	15/23 (65.2%; 42.7%-83.6%)	10/13 (76.9%; 46.2%-95.0%)	491/814 (60.3%; 56.9%-63.7%)
R-G+	3/15 (20.0%; 4.3%-48.1%)	1/10 (10.0%; 0.25%-44.5%)	77/491 (15.7%; 12.6%-19.2%)
R-C+	2/15 (13.3%; 1.7%-40.5%)	2/10 (20.0%; 25.2%-55.6%)	106/491 (21.6%; 18.0%-25.5%)
Syphilis Serology Tested	21/23 (91.3%; 72.0%-98.9%)	13/13 (100%)	814/814 (100%)
Acute	6/21 (28.6%; 11.3%-52.2%)	5/13 (38.5%; 13.9%-68.4%)	85/814 (10.4%; 8.4%-12.8%)
Old/Chronic	7/21 (33.3%; 14.6%-57.0%)	7/13 (53.9%; 25.1%-80.8%)	401/814 (49.3%; 45.8%-52.8%)
Non-Reactive	8/21 (38.1%; 18.1%-61.6%)	1/13 (7.7%; 0.2%-36.0%)	316/814 (38.8%; 35.5%-42.3%)

	Screening HCV Ab Performed?	Odds Ratio (95% CI)	P value
Baseline HCV- with Pharyngeal G/C Performed	413/500 (82.6%; 79.0%-85.8%)	2.78 (2.01-3.85)	<.001
Baseline HCV- without Pharyngeal G/C Performed	198/314 (63.1%; 57.5%-68.4%)		
Baseline HCV- with Rectal G/C Performed	401/491 (81.7%; 78.0%-85.0%)	2.40 (1.74-3.31)	<.001
Baseline HCV- without Rectal G/C Performed	210/323 (65.0%; 59.5%-70.2%)		
Baseline HCV- with Pharyngeal or Rectal G/C Performed	423/521 (81.2%; 77.6%-84.5%)	2.41 (1.74-3.34)	<.001
Baseline HCV- without Pharyngeal or Rectal G/C Performed	188/293 (64.2%; 58.4%-69.7%)		

	Age <=35 (n=304)	Age >35 (n=572)	Odds Ratio (95% CI)	P value
Urine G/C Tested	299/304 (98.4%; 96.2%-99.5%)	490/572 (85.7%; 82.5%-88.4%)	10.01 (4.01-24.97)	<.001
U-G+	32/299 (10.7%; 7.4%-14.8%)	16/490 (3.3%; 1.9%-5.3%)	4.09 (2.21-7.58)	<.001
U-C+	28/299 (9.4%; 6.3%-13.3%)	10/490 (2.0%; 0.9%-3.7%)	5.70 (2.73-11.91)	<.001
Pharyngeal G/C Tested	241/304 (79.3%; 74.3%-83.7%)	294/572 (51.4%; 47.2%-55.6%)	3.62 (2.62-4.99)	<.001
P-G+	57/241 (23.7%; 18.4%-29.5%)	21/294 (7.1%; 4.5%-10.7%)	6.06 (3.59-10.21)	<.001
P-C+	18/241 (7.5%; 4.5%-11.6%)	8/294 (2.7%; 1.2%-5.3%)	4.44 (1.91-10.33)	<.001
Rectal G/C Tested	241/304 (79.3%; 74.3%-83.7%)	282/572 (49.3%; 45.1%-53.5%)	3.93 (2.85-5.43)	<.001
R-G+	53/241 (22.0%; 16.9%-27.8%)	28/282 (9.9%; 6.7%-14.0%)	4.10 (2.53-6.64)	<.001
R-C+	73/241 (30.3%; 24.6%-36.5%)	37/282 (13.1%; 9.4%-17.6%)	4.57 (2.99-6.99)	<.001
Syphilis Serology Tested	301/304 (99.0%; 97.1%-99.8%)	555/572 (97.0%; 95.3%-98.3%)	3.07 (0.89-10.57)	.061
Acute	54/301 (17.9%; 13.8%-22.8%)	43/555 (7.8%; 5.7%-10.3%)	2.66 (1.73-4.08)	<.001
Old/Chronic	134/301 (44.5%; 38.8%-50.3%)	291/555 (52.4%; 48.2%-56.7%)	0.76 (0.58-1.01)	.055
Non-Reactive	113/301 (37.5%; 32.1%-43.3%)	221/555 (39.8%; 35.7%-44.0%)	0.94 (0.71-1.25)	.671

	NH-Black (n=415)	NH-White (n=125)	Hispanic (n=290)	Odds Ratio (95% CI)	P value
Urine G/C Tested	380/415 (91.6%; 88.5%-94.1%)	114/125 (91.2%; 84.8%-95.5%)	254/290 (87.6%; 83.2%-91.2%)	1.38 (0.88-2.17)	.160
U-G+	36/380 (9.5%; 6.7%-12.9%)	4/114 (3.5%; 1.0%-8.7%)	6/254 (2.4%; 0.9%-5.1%)	3.55 (1.82-6.93)	<.001
U-C+	28/380 (7.4%; 5.0%-10.5%)	3/114 (2.6%; 0.6%-7.5%)	7/254 (2.8%; 1.1%-5.6%)	3.26 (1.57-6.80)	.001
Pharyngeal G/C Tested	269/415 (64.8%; 60.0%-69.4%)	74/125 (59.2%; 50.1%-67.9%)	163/290 (56.2%; 50.3%-62.0%)	1.35 (1.03-1.78)	.031
P-G+	50/269 (18.6%; 14.1%-23.8%)	6/74 (8.1%; 3.0%-16.8%)	16/163 (9.8%; 5.7%-15.5%)	2.12 (1.31-3.43)	.002
P-C+	14/269 (5.2%; 2.9%-8.6%)	3/74 (4.1%; 0.8%-11.4%)	8/163 (4.9%; 2.1%-9.4%)	1.31 (0.60-2.86)	.502
Rectal G/C Tested	264/415 (63.6%; 58.8%- 68.3%)	72/125 (57.6%; 48.4%-66.4%)	161/290 (55.5%; 49.6%-61.3%)	1.36 (1.04-1.79)	.025
R-G+	43/264 (16.3%; 12.1%-21.3%)	13/72 (18.1%; 10.0%-28.9%)	19/161 (11.8%; 7.3%-17.8%)	1.29 (0.81-2.03)	.280
R-C+	71/264 (26.9%; 21.6%-32.7%)	10/72 (13.9%; 6.9%-24.1%)	26/161 (16.2%; 10.8%-22.8%)	2.23 (1.47-3.39)	<.001
Syphilis Serology Tested	406/415 (97.8%; 95.9%-99.0%)	123/125 (98.4%; 94.3%-99.8%)	281/290 (96.9%; 94.2%-98.6%)	1.10 (0.45-2.69)	.830
Acute	52/406 (12.8%; 9.7%-16.5%)	17/123 (13.8%; 8.3%-21.2%)	24/281 (8.5%; 5.6%-12.4%)	1.32 (0.87-2.02)	.192
Old/Chronic	209/406 (51.5%; 46.5%-56.4%)	56/123 (45.5%; 36.5%-54.8%)	133/281 (47.3%; 41.4%-53.4%)	1.15 (0.88-1.50)	.300
Non-Reactive	145/406 (35.7%; 31.1%-40.6%)	50/123 (40.7%; 31.9%-49.9%)	124/281 (44.1%; 38.2%-50.2%)	0.77 (0.59-1.02)	.065

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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

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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 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 HBV 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-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.

Figure 1. Flow chart of inclusion and exclusion of subjects
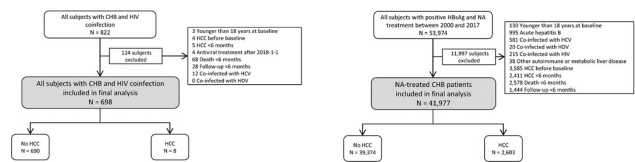
692 patients who had all available laboratory parameters were included in the propensity score.

38,102 patients who had all available laboratory parameters were included in the propensity score.

Figure 2. Kaplan Meier curves showing incidence of hepatocellular carcinoma in HBV/HIV co-infected and HBV mono-infected patients
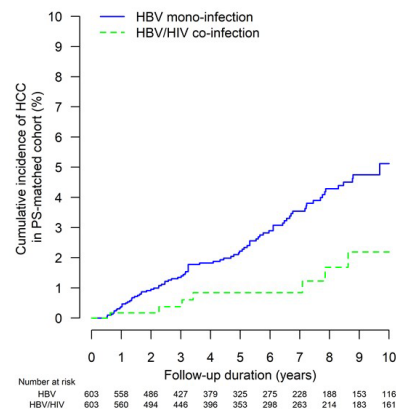
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359. TLR7 Gene Polymorphisms Influence Development of Hepatic Fibrosis in HCV/HIV Coinfection

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Background. Hepatic fibrosis in individuals with HCV/HIV coinfection or HCV mono-infection may be modulated by a variety of host factors. In this study, we investigated the role of gene polymorphisms of putative genes that might influence fibrosis progression including MICA (rs2596542), interferon-stimulated gene OAS2 (rs1293762) and the pathogen recognition receptors TLR7 (rs179009) and TLR9 (rs187084). Effect on a cytokine panel was evaluated.

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

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 Statistix 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 TLR7 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 TLR7 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 TLR7 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 TLR7 A -> G polymorphism, while RANTES levels were inversely associated (decreased, $P = 0.0443$) with it.

Conclusion. Of the gene polymorphisms investigated, only TLR7 (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.

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

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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. Variables included demographics, HIV risk factors, comorbidities, HBsAg loss, HepBeAg, CD4+ count, HBV DNA, and HIV RNA viral load. Bivariate analysis was performed using chi-square and student t-test as appropriate; a logistic regression model was used to identify independent associations among significant variables (STATA).

Results. Within those with HIV/HBV coinfection ($n = 501$), 89 (18%) met the criteria for ALD (92% male, 47% Black, 33% White, 16% Hispanic, 73% >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 ($P \leq 0.001$). 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, HepBeAg status or baseline CD4+ count, HBV DNA, HIV RNA, and AIDS.

Conclusion. Increased monitoring for the presence of ADL should be conducted in HIV/HBV coinfection. Particular attention and surveillance should be paid to those with 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 ADL in HIV/HBV patients.

Table 1. Logistic Regression Analysis

Variables	Adjusted Odds Ratio	95% CI	P value
Race/Ethnicity			
White	Reference		
Black	0.51	0.29, 0.90	0.02
Hispanic	0.73	0.34, 1.55	0.41
Other	1.15	0.33, 3.98	0.82
Age group			
< 30 years	Reference		
30-39 years	1.46	0.46, 4.60	0.52
40-49 years	2.87	0.96, 8.63	0.06
>50 years	3.79	1.16, 12.38	0.03
Diabetes	1.98	1.02, 3.83	0.04
Hepatitis C	3.2	1.72, 5.94	<.001

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

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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 inadvertently causing re-emergence of lamivudine-resistant HBV is present. We report two HIV patients with residual whole-genome HBV with lamivudine and telbivudine 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 telbivudine, 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.

Table 1. Patient characteristics and viral mutations.

	Case 1: 22/M	Case 2: 42/M
Baseline CD4 count (cells/mm³)	269	45
Nadir CD4 count (cells/mm³)	7	45
Baseline HIV viral load (copies/mL)	101,000	97,000
HIV genotype	PR: CRF01_AE RT: CRF01_AE	PR: CRF01_AE RT: CRF01_AE
HIV mutations	NRTIs: K65R NNRTIs: G190A, Y181C, Y188L PIs: none	NRTIs: K65R, D67N, M184I NNRTIs: G190Q PIs: none
Initial ART Regimen	tenofovir/lamivudine/efavirenz	tenofovir/lamivudine/efavirenz
Revised ART Regimen	zidovudine/lamivudine/lopinavir-ritonavir	zidovudine/lamivudine/lopinavir-ritonavir
HBV viral load (copies/mL, limit of detection is 179)	undetectable	undetectable
HBV Genotype	A	A
HBV mutations	L180M, M204, L223M, M247V	L180M, M204, L223M,M247V

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

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Background. HIV and HCV infection are emerging global public health problems. People living with untreated HIV 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 this 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