Figure 3. Associations between alcohol use and SVR12 by HIV status



Abbreviations: referent group = abstinent; AUD = alcohol use disorder Notes: modeled separately by HIV status; adjusted for age, FIB-4, body mass index, and hepatic decompensation

Disclosures. All authors: No reported disclosures.

2225. Acute Hepatitis C Virus Infections in HIV-Infected Persons in the Era of Direct-Acting Antiviral Therapy

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Background. Acute hepatitis C (HCV) infection can be sexually transmitted in HIV-infected men who have sex with men (MSM). Since 2014, direct-acting antivirals (DAA) have successfully cured many persons with chronic HCV infection. We examined the incidence of acute HCV infection in HIV-infected persons before and after the widespread use of DAA therapy.

Methods. We used the HIV Atlanta Veterans Affairs Cohort Study (HAVACS) to examine the incident rate (IR) of acute HCV infections during the period of 01 January 2013 to 31 December 2013 (pre-DAA era) and in the post-DAA era (January 1, 2017 to December 31, 2017). Acute HCV infection was identified using HCV seroconversion or HCV viremia with a negative HCV antibody. We also describe the demographic, clinical characteristics, and virologic outcomes of acute HCV infection cases observed since 2014.

Results. In the pre-DAA era, 56 cases of acute HCV were seen among 1,378 persons (IR: 40.6 per 1000). In the post-DAA era, 29 cases were seen among 1,433 persons (IR: 20.2 per 1,000). HAVACS persons seen in 2017 were 52% less likely to be diagnosed with acute HCV infection than those seen in 2013. Of the seven acute HCV cases examined in detail, the median age is 41 years (range 33–60 years). All cases were male and African American race. Two persons had active IV drug use in addition to unprotected anal intercourse as a risk factor for HCV infection. The median CD4 just prior to HCV infection was 753 cells/cm² (range: 590–1,046 cells/cm²). One person had a detectable HIV viral load (527 copies/mL) just prior to HCV infection while the other 6 persons had undetectable HIV viral loads. The peak AST ranged from 147 to 1,256 IU/L (median: 798 IU/L) while the peak ALT ranged from 171 to 1,530 IU/L (median: 855 IU/L). The median total bilirubin is 3.5 mg/dL. One person spontaneously cleared his HCV infection, two were treated with DAA therapy, and the other four are under active monitoring.

Conclusion. Acute HCV infections have significantly decreased in HIV-infected persons in the DAA era. However, acute HCV infections can cause severe transaminitis and jaundice. More work is needed to prevent HCV infections in HIV-infected persons.

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2226. Immune Checkpoint Inhibitors in Solid Tumor Patients with Chronic Hepatitis C Virus Infection: A Prospective Case-Series

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Background. Immune checkpoint inhibitors are a novel class of targeted therapy that activates T cell-mediated tumor cell death. Controversies exist about the safety and

efficacy of immunotherapy in patients with chronic viral infections affecting T cells, such as hepatitis C virus (HCV). Herein, we analyzed the effect of immune checkpoint inhibitors on HCV viremia and HCV-related hepatic outcome.

Methods. HCV-infected patients with solid tumors seen at MD Anderson Cancer Center (November 2012–April 2018) were enrolled in a prospective observational study. Patients were monitored for the development of HCV reactivation (HCV-RNA $\geq 1 \log_{10}$ IU/mL over baseline), hepatitis flare (alanine transaminase increase to ≥ 3 times upper limit of normal) and HCV-associated hepatitis (HCV reactivation and hepatitis flare) while on cancer treatment.

Results. Out of 205 chronically infected patients with solid tumors, 12 (6%) received immunotherapy and were seen in the HCV clinic, but only four (2%) returned for regular monitoring (Table 1). They were followed for 9 months. None of the four patients received concomitant chemotherapy or steroids. Hepatitis flare occurred in three patients, but HCV reactivation or HCV-associated hepatitis was not detected in any study patient. Immune checkpoint inhibitors were discontinued in one patient (25%) due to hepatitis flare unrelated to HCV.

Conclusion. The use of immune checkpoint inhibitors appears to be safe in solid tumor patients with HCV infection.

 Table 1.
 Demographics, Types of Cancer, Immunotherapy Received, and Changes in Serum HCV-RNA

								Highest	
							Baseline HCV-RNA	HCV-RNA After Baseline	
Patient	Age, years		HCV Genotype	Cirrhosi	s Type of Cancer	Immunotherapy	(log10IU/ mL)	(log10IU/ mL)	Hepatitis Flare
1	67	Male	1a	Yes	Hepatocellular carcinoma	Nivolumab + ipilimumab	5.45	5.08	Yes ^a
2	52	Male	1a	No	Melanoma	Nivolumab + ipilimumab	6.97	6.95	Yes ^b
3	57	Male	3	No	Melanoma	Pembrolizumab	5.72	6.39	No
4	55	Male	1a	No	Melanoma	Ipilimumab	5.75	7.18 °	Yes ^c

^a After partial hepatectomy

^b Negative infectious work-up including hepatitis A, B, E, cytomegalovirus, and herpes simplex virus.

^c Six months after completing immunotherapy, HCV-associated hepatitis occurred after starting high-dose steroids for brain metastasis.

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2227. Short-Duration of Direct-Acting Antivirals in Hepatitis C Virus-Infected Cancer Patients

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Background. Short-duration with an 8-week course of ledipasvir/sofosbuvir (LDV/SOF) or glecaprevir/pibrentasvir (GLE/PIB) is considered adequate to treat hepatitis C virus (HCV) infection in selected patients. However, immunocompromised patients with HCV/HIV are not eligible for this approach. Herein, we study the efficacy and safety of an 8-week therapy with direct-acting antivirals (DAAs) in HCV-infected cancer patients.

Methods. HCV-infected patients with any type of cancer followed at MD Anderson Cancer Center (June 2014–April 2018) and treated with an 8-week course of LDV/SOF or GLE/PIB were enrolled in a prospective observational study. Efficacy was calculated based on achieving sustained virologic response at 12 weeks (SVR12) after end of treatment per intention to treat (ITT) analysis. A posthoc per-protocol (PP) analysis was done in patients with 12 weeks of follow-up post DAAs. Safety was assessed by emergence of adverse events (AEs) and clinically significant drug–drug interactions (DDIs).

Results. Twenty-four patients were treated with a short-duration of DAAs, 22 with LDV/SOF and two with GLE/PIB. General characteristics are described in Table 1. Five patients received concomitant cancer treatment (nivolumab, sorafenib, lenalidomide, tamoxifen and leuprolide), without DDIs noted. Among the patients who have completed DAAs, SVR rates were 87% per ITT (20/23) and 100% PP (20/20) analyses. No patients had grade 2, 3 or 4 AEs.

Conclusion. This is the first prospective study to evaluate the use of short-duration of DAAs in HCV-infected cancer patients where these regimens were found to be effective and safe.

Table 1. General Characteristics

Characteristics	Patients <i>N</i> (%) 24		
Number of patients			
Age, median (interquartile range)	61 (57–66)		
Male sex	18 (75)		
Black race	9 (38)		
Obesity (body mass index >30)	10 (42)		
HCV genotype			
1a	17 (71)		
1b	5 (21)		
2	2 (8)		
Type of cancer			
Hematologic ^a	6 (25)		
Solid ^b	18 (75)		

^a Multiple myeloma (2), acute myeloid leukemia (2), non-Hodgkin lymphoma (2).

^b Prostate (3), head and neck (3), lung (3), renal (2), anal (2), ovarian (2), breast (1), thyroid (1), gastrointestinal stromal tumor (1).

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2228. Late Viral Relapse After Direct-Acting Antiviral Treatment in Hepatitis C Virus-Infected Cancer Patients

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Background. According to professional societies, the endpoint to consider hepatitis c virus (HCV) infection cured is the achievement of a sustained virologic response

12 weeks after treatment completion (SVR12). Late recurrences (beyond SVR12) are rare. Herein, we report two cases of HCV-infected cancer patients with late relapses post direct-acting antivirals (DAAs).

Methods. Patients with any type of chronic cancer and HCV treated with DAAs between January 2014 and March 2018 at MD Anderson Cancer Center were prospectively followed. All patients had HCV RNA levels at baseline; 2 and 4 weeks after initiation of DAAs; at end of treatment (EOT); and 12 weeks after completion of DAAs. No phylogenetic analyses were available for samples collected.

Results. Among 196 HCV-infected cancer patients treated with DAAs, 20 developed viral relapse, 2 (10%) of them with late relapse (Figure 1). Both patients denied behaviors, exposures, and conditions associated with HCV reinfection. Case 1: Fifty-six-year-old male with hepatocellular carcinoma (HCC), HCV genotype 1a, interferon-experienced, with compensated cirrhosis received in 2017 ledipasvir/sofosbuvir for 12 weeks, followed by systemic chemotherapy with sorafenib. He achieved an SVR12 but developed HCV relapse 12 weeks later (24 weeks after EOT). Patient remained infected with HCV 1a. He did not receive retreatment due to HCC not amenable for curative treatment. Case 2: 57-year-old male with multiple myeloma, HCV genotype 1a, interferon-experienced without cirrhosis. He received sofosbuvir and simeprevir in 2015 for 12 weeks. Post DAAs, he received chemotherapy with carfilzomib, lenalidomide, dexamethasone, and ixazomib followed by autologous hematopoietic cell transplant pre-conditioned with melphalan. He achieved both an SVR12 and SVR 24 but had HCV relapse detected during the one year follow-up visit. Patient remained infected with HCV 1a. He has retreated with sofosbuvir, veltapasvir, voxilaprevir and ribavirin and currently with HCV RNA level at EOT.

Conclusion. Late HCV relapses can occur in HCV-infected cancer patients. Long-term monitoring of HCV-RNA and easy-to-use tests to differentiate relapses from reinfection in real-world practice are warranted in this population.

Figure 1. HCV-infected cancer patients with late viral relapse. Abbreviations: SVR12, sustained virologic response 12 weeks after treatment completion: SVR24, sustained virologic response 24 weeks after treatment completion.



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2229. Low Hepatitis C Virus Reinfection Rates After Sustained Viral Response in HIV Co-infected Patients in Houston, Texas

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Background. Hepatitis C Virus (HCV) infection is a significant public health problem associated with a high morbidity and mortality. HCV recurrence is a particular concern in patients with ongoing high-risk behaviors. Previous studies have shown a wide variation in HCV reinfection rates, but have considered small selected populations. The aim of our study was to estimate the HCV reinfection rates in a representative real-world cohort of HCV/HIV co-infected patients in Houston, Texas and to compare it with published data.

Methods. Retrospective cohort study of HCV/HIV co-infected patients treated between January 2004 to July 2016 at a freestanding HIV clinic that serves indigent and minority patients. HCV reinfection was defined as a single detectable HCV RNA level after achieving SVR 12. We reviewed demographic data, risk behaviors, laboratory tests and treatment outcomes. Cox proportional hazards regression was used to estimate reinfection rates. A meta-analysis was performed to calculate the reinfection rates reported in the literature in different patient populations.

Results. Of 288 patients treated, 187 (65%) achieved SVR12 by the end of the study. Follow-up data were available in 151 (81%) patients. Median follow-up time after SVR12 was 1.26 (0.66, 2.13) years. After achieving SVR12, two patients became reinfected, with a reinfection rate of 10.8 (1.3–39.1) per 1,000 PYFU. Our meta analysis demonstrated higher reinfection rates in different populations (87.8 (60.9–127) per 1,000 PYFU in MSM; 65.6 (34.1–126) per 1,000 PYFU and 13.5 (10.4–17.5) per 1,000 PYFU in non-IVDU). In our patient population, the mean time from SVR12 to reinfection was 52.5 weeks, and reinfection was with the same HCV genotype. Both patients were MSM and reported high-risk sexual behavior; one patient also developed syphilis. Both patients have been retreated. One has achieved SVR12 and the following weeks.

Conclusion. The reinfection rate in our diverse cohort of HIV/HCV treated patients is very low compared with others studies. Efforts to reduce risk behaviors are important if HCV elimination is to be achieved.

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2230. Treatment Outcomes for Hepatitis C Patients from Two Federally Qualified Community Health Centers in South Carolina

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Background. Approximately 3.9 million Americans live with chronic Hepatitis C virus (CHCV). Major advances have been made in the treatment of CHCV, with the availability of oral directly acting antiviral (DAA) regimens. However, significant barriers to treatment remain for patients accessing safety net providers for care. In 2011, 61,294 Community Health Center (CHC) patients had Hepatitis C as their primary diagnosis. This study provides insight into unique CHC patient characteristics and outcomes of care at two federally qualified health centers (FQHC).

Methods. We queried electronic health records (EHR) from Q4 2014 to Q1 2018 for Hep C patients attending two FQHCs in South Carolina (n = 223). Data from both practices were aggregated to capture sustained virologic (SVR) rates at 12 weeks post treatment. Patient demographic factors, including age; gender; race/ethnicity, insurance status and people who inject drugs (PWID) were extracted. Clinical measures such as baseline and post treatment viral loads, Fibrosure, AST to Platelet Ratio Index (APRI) measures, pre treatment and post treatment liver ultrasound screening, HCV genotype, and HIV co-infection are reported. Patient outcomes were monitored using SVR viral load values (detectable or nondetectable) at 12 weeks and 1 year from treatment onset.

Results. Mean age was 57.03 SD \pm 0.65 with 71.7% of the population treated aged 55 or older. Most patients were males (63.2%), African American (68.2%) and uninsured (31.4%). Median baseline HCV viral load was 1,950,000 IU/mL. About 95.9% of the patients were naïve to Hepatitis C treatment. Majority of Fibrosure stages (F0–F2 48.9%; and F3–F4 37.2%) and APRI scores both showed about half of patients presented with little likelihood of liver cirrhosis. Post-liver ultrasound occurred in 37.7% of the population. Top three genotypes were 1a (67.3%), 1b (17.5%) and 2b (5.8%). The proportion of PWID among those responding was 23.4%.HIV coinfection in the population sample was 29.1%, while the SVR VL was nondetectable for 97.6%.