

302. HCV Care in Federally Qualified Health Centers During the Opioid Epidemic: A Retrospective Cohort Study

Sabrina A. Assoumou, MD, MPH¹; Jianing Wang, MSc²; Shayla Nolen, MPH³; Golnaz Eftekhari Yazdi, MSc²; Kenneth H. Mayer, MD, MPH³; Jon Puro, MPA/HA⁴; Joshua A. Salomon, PhD⁵ and Benjamin P. Linas, MD, MPH¹; ¹Boston University School of Medicine/Boston Medical Center, Wayland, Massachusetts; ²Boston Medical Center, Boston, Massachusetts; ³Harvard Medical School/Fenway Research Institute, Boston, Massachusetts; ⁴OCHIN, Inc., Portland, Oregon; ⁵Stanford School of Medicine, Stanford, California

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Background. Federally qualified health centers (FQHCs) serve diverse communities in the United States (US) and could function as important venues to manage hepatitis C virus (HCV) infections. Little is known on HCV outcomes in underserved communities as most of the current data are derived from clinical trials, commercially insured patients, or small health center samples. We aimed to determine the proportion of HCV testing, factors associated with treatment initiation, and real-world treatment outcomes in a large, national diverse sample of US FQHCs during the opioid epidemic.

Methods. We created a retrospective cohort of adults seen at 341 participating FQHCs in 19 US states. Inclusion criteria were: (1) clinical visit between January 01, 2012 and June 30, 2017; (2) ≥18 years of age. Outcomes included HCV testing proportion, stratified by diagnosis of opioid use disorder (OUD); treatment initiation rates; and sustained virologic response (SVR), defined as undetectable HCV RNA 3 months after treatment completion. We identified factors associated with testing, treatment initiation, and SVR using logistic regression.

Results. Of the 1,508,525 patients meeting inclusion criteria, 88,384 (5.9%) were tested for HCV, and 8,694 (9.8%) of individuals tested had reactive results. Of the 6,357 with HCV RNA testing, 4,092 (64.4%) had detectable RNA. Twelve percent of individuals with chronic HCV and evaluable data initiated treatment. Of those, 86% reached SVR. Having commercial insurance (aOR, 2.10, 95% CI, 1.45–3.02), older age (aOR, 1.07, 95% CI, 1.06–1.09) and being Hispanic/Latino (aOR, 1.35, 95% CI, 1.33–1.38) or Asian/Pacific Islander (aOR, 1.84, 95% CI, 1.79–1.90) were independently associated with higher odds of treatment initiation after multivariable adjustment. Only 8% of individuals with chronic HCV were tested for HIV, and 15% of individuals with identified OUD were tested for HCV.

Conclusion. During the opioid epidemic, fewer than 20% of individuals with identified OUD were tested for HCV at evaluated FQHCs. In addition, approximately 10% of patients initiated treatment and SVR was lower than expected. Expansion of HCV management into community clinics must consider measures to monitor and evaluate treatment effectiveness, and to improve outcomes if cure rates are low.

Disclosures. All authors: No reported disclosures.

303. A Surrogate Rodent Model for Studying Hepatitis C Virus-specific CD8 T-cell Impairment and Vaccine Prevention

Alex S. Hartlage, BS¹ and Amit Kapoor, PhD²; ¹The Ohio State University College of Medicine, Dublin, Ohio; ²Nationwide Children's Research Institute, Columbus, Ohio

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Background. Virus-specific CD8 T cells are essential for control of acute hepatitis C virus (HCV) infections, yet spontaneously fail in most patients leading to lifelong chronicity and increased risk for severe liver diseases. Efforts to study HCV-specific CD8 T-cell impairment have been hampered by a lack of small animal models. Recently, we established a rat model of chronic HCV-like infection using a hepatitis virus homolog identified in *Rattus norvegicus*. The nature of virus-specific CD8 T-cell immunity in this model has yet to be determined.

Methods. Using two MHC class I tetramers against epitopes located in the E1 and NS5B proteins, we tracked the induction and phenotype of virus-specific CD8 T cells during chronic infection. Responses to infection were similarly analyzed in immune rats that had been vaccinated against the NS3-5B proteins, a strategy that is effective in this experimental setting.

Results. Virus-specific CD8 T cells expanded vigorously in liver shortly after infection but did not develop into functional effectors based upon failure to produce cytokines (IFN γ , TNF α , IL-2, IL-4, IL-10, IL-17A) following peptide stimulation. Notably, subversion of responses was not due to viral escape from T-cell recognition, but rather an intrinsic defect in the antiviral response. Indeed, these populations expressed the inhibitory receptor programmed cell death-1 and other markers consistent with an arrested effector-like state precluded from long-term memory formation (CD127^{CD27}CD28^{CD62L}GranzymeB⁺). In contrast, adenoviral immunization of naïve rats protected virus-specific T cells from functional impairment after infection and supported memory response development, including against the E1 epitope not encoded by vaccine.

Conclusion. Together, our findings reveal a spontaneous failure of virus-specific CD8 T cells following rat hepatitis virus challenge that is highly reminiscent of human HCV infections. Furthermore, these results highlight the utility and significance of this model for understanding mechanisms of HCV persistence and protective immunity necessary for the development of effective vaccines and immune interventions.

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304. Hepatocellular Carcinoma Occurs Frequently and Early After Treatment in HCV Genotype 3 Infected Persons Treated with DAA Regimens

Ghias Un Nabi Tayyab, MBBS¹; Shafqat Rasool, MBBS¹; Bilal Nasir, MBBS¹ and Adeel A. Butt, MD, MS²; ¹Post Graduate Medical Institute/Ameer Ud Din Medical College/Lahore General Hospital, Lahore, Punjab, Pakistan; ²Weill Cornell Medical College, Doha, Ad Dawhah, Qatar

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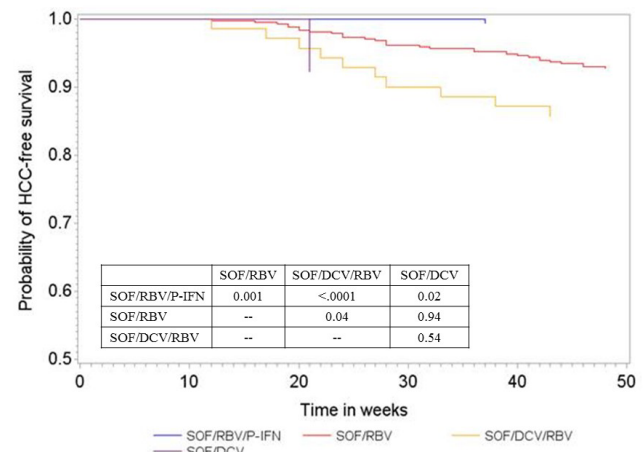
Background. Treatment of HCV with directly acting antiviral agents (DAAs) is associated with a significant reduction in cardiovascular, metabolic and cancer risk. However, there are conflicting data regarding the risk of hepatocellular carcinoma (HCC) after DAA treatment. Risk of HCC in HCV genotype 3 infected persons after DAA therapy is not well known.

Methods. We prospectively studied HCV-infected persons initiated on treatment between October 2014 and March 2017 at two centers in Pakistan. All persons were free of HCC at study initiation. The occurrence of HCC was confirmed based on radiologic findings on a triphasic CT on 64 slice MDCT scanner. The treatment regimen was at the discretion of clinical care providers, taking into account the national guidelines and patient preferences. Patients were followed for 24 weeks after the completion of therapy. Informed consent was obtained from all participants.

Results. A total of 662 persons were initiated on treatment. Median age (IQR) was 50 (41, 57) years and 48.8% were male. At baseline, 49.4% were cirrhotic with 90% of cirrhotics having compensated cirrhosis. 91% were genotype 3 and SVR was attained in 91.9%. Treatment regimens used were: Sofosbuvir (SOF)/ribavirin (RBV)/pegylated interferon (PEG-IFN), 25.2%; SOF/RBV, 62.4%; SOF/RBV/daclatasvir (DCL), 10.6%; SOF/DCL, 2.0%. Incident HCC was detected in 42 patients (12.8%) in the six month period after treatment completion, and was exclusively observed in those with cirrhosis. In multivariable Cox regression analysis, SVR was associated with a reduction in HCC risk (HR, 95% CI: 0.35, 0.14, 0.85) while SOF/RBV/DCL regimen (compared with SOF/RBV/PEG-IFN) was associated with an increased risk of HCC (HR, 95% CI: 17.32, 2.14, 140.36). In K-M plots by treatment regimen, those treated with SOF/RBV, SOF/RBV/DCL, or SOF/DCL regimens had shorter HCC-free survival compared with those treated with a SOF/RBV/PEG-IFN regimen. (See figure)

Conclusion. In a predominantly genotype 3 cohort, incident HCC occurs commonly and early after treatment completion, and exclusively in those with pretreatment cirrhosis. SVR reduces but does not completely eliminate the risk of HCC. Treating HCV-infected persons before the development of cirrhosis may reduce future risk of HCC.

Figure. Hepatocellular carcinoma free survival, by treatment regimen



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305. Using Big Data to Re-Engage Hepatitis C-infected Persons: A UK Operational Delivery Network's Experience

William Osborne, MBBS, MRCP, BSc, DTM&H¹; Noorann Sheikh, MBBS²; Andrew Bosworth, PhD³; Sally Bufton, BSN⁴; David Mutimer, MBBS, MD⁵; Mamoona Tahir, MBBS⁵ and Sowsan F. Atabani, MBBS⁵; ¹Royal Stoke and University Hospital, Stoke-on-Trent, UK; ²University Hospitals Birmingham, Birmingham, UK; ³Public Health England Birmingham Laboratory, National Infection Service, Public Health England, Birmingham, UK; ⁴University Hospitals Birmingham, UK, Birmingham, UK; ⁵Public Health England, Birmingham, UK

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Background. The prevalence of hepatitis C (HCV) varies across different risk groups in the UK. In England, responsibility for the co-ordination and administration of DAAs (direct-acting antivirals) to HCV PCR positive patients is with 22 regional