Table 2: On treatment % with decline in eGFR and % developing grade 3/4 anemia in persons treated with solofabuvir/sofosbuvir or paritaprevir/ritonavir/ombitasvir + dasabuvir regimens

| Baseline eGFR | SOLO/DV, no RBV | a | SOLO/DV+RBV | b | P-value (SOLO/DV vs. SOLO/DV+RBV) |
|---------------|----------------|---|-------------|---|----------------------------------|
| CKD Stage 1   |                | 12.4% | 0.01 | 23.9% | 0.01 |
| CKD Stage 2   |                | 12.3% | 0.01 | 23.9% | 0.01 |
| CKD Stage 3   |                | 12.3% | 0.01 | 23.9% | 0.01 |
| CKD Stage 4   |                | 12.3% | 0.01 | 23.9% | 0.01 |

A. Discussion. The primary outcome was HCV testing by Medicaid from 10/1/15 to 9/30/16. The primary outcome was HCV testing by PCR (CPT 87520 [HCV, direct probe], 87521 [HCV, amplified probe], 87522 [HCV RNA, Quantitative]) or antibody (CPTs 86803-4). Testing for HIV (CPTs 86701, 86702, 87523, 87525, 87526) and syphilis (CPT 86592) was also recorded. NAS was defined as presence of ICD-10-CM code P96.1 in any diagnosis field. Descriptive statistics were used.

Results. During the study period, 625 children born with 4055 [median, Interquartile range (IQR) 1–8] claims were HCV-exposed. The majority of children were white (393, 60%), non-Hispanic (420, 67%) and male (318, 51%). Patterns of testing are shown in the Table.

Table: Medicaid claims for tests performed in children perinatally exposed to HCV

| Test               | Number of Children | Median Age (months) |
|--------------------|--------------------|---------------------|
| HCV PCR            | 69 (11)            | 3 [2, 6]            |
| HCV antibody       | 11 (2)             | 6 [2, 8]            |
| HIV PCR            | 30 (6)             | 2 [2, 3]            |
| HIV antibody       | 8 (1)              | 2 [2, 3]            |
| Syphilis           | 26 (4)             | 2 [2, 3]            |

*a Child may have been tested for more than one infection during the study period.

Conclusion. The proportion of HCV-exposed infants with a claim for HCV testing is low in the KY Medicaid population; testing for other perinatally-acquired infections is even less common. Children with NAS were less likely to be tested. Statewide guidelines for appropriate testing in children with perinatal HCV exposure and NAS are urgently needed.

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52. Fibrosis Progression and Incidence of Cirrhosis and Hepatic Decompensation in Persons Treated with Paritaprevir/Ritonavir/Ombitasvir/ Dasabuvir: Results from ERCHIVES

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Background. Data are limited regarding the effect of paritaprevir/ritonavir, ombitasvir, dasabuvir regimen (PrOD) upon the rate of liver fibrosis progression and incidence of cirrhosis and hepatic decompensation after treatment for HCV.

Methods. Within ERCHIVES (Electronically Retrieved Cohort of HCV Infected Veterans), we identified HCV infected persons treated with PrOD and treatment-naive controls to determine the effect of PrOD treatment upon subsequent progression of fibrosis and incident cirrhosis and hepatic decompensation. Controls were propensity-score matched based on demographic and clinical characteristics. We excluded those with HIV coinfection, positive HBsAg, hepatocellular carcinoma at baseline and those with missing HCV RNA or FIB-4 scores. Fibrosis progression and liver cirrhosis were assessed using the FIB-4 score.

Results. The final propensity score matched sample included 1,473 PrOD-treated individuals, and an equal number of matched, untreated controls. PrOD-treated patients had significantly reduced median FIB-4 scores over time, compared with controls (median absolute change in FIB-4 = -0.7 [IQR -1.51, -0.3] vs. +0.06 [IQR -0.38, 0.49], P < 0.0001). Compared with matched controls, PrOD-treated patients had an 86% relative reduction in the risk of incident cirrhosis over 2,241 patient-years of follow-up (adjusted HR 0.14 [95% CI 0.08-0.23]). Treatment with PrOD was also associated with delayed time to first hepatic decompensation event (P < 0.001). In sensitivity analysis, the exclusion of patients with baseline cirrhosis did not materially alter the estimates of effect.

Conclusion. Treatment with PrOD is associated with a significant reduction in fibrosis progression, a longer time to the development of cirrhosis, and reduced risk of