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## "Delays in Hepatitis C Fibrosis Staging on Liver Function"

Hepatitis C virus (HCV) claims more American lives than the next 60 reportable infectious diseases combined.<sup>1</sup> Our study objective was to assess delays in hepatic fibrosis staging on decline in liver function. We undertook a retrospective cohort study of all individuals diagnosed with HCV by UMCNO ED between March 1, 2015 and August 1, 2017 and who received subsequent hepatic fibrosis staging. Data was collected by electronic chart review. Exposure was defined as time from chronic HCV diagnosis to fibrosis staging. Our primary outcome was change in liver function measured by AST to Platelet Ratio Index (APRI) and Fibrosis-4 (FIB4) Index. Measures of hepatic function were collected at time of HCV screening, time of fibrosis staging, and start of HCV therapy (if achieved). A secondary analysis was conducted among patients who initiated treatment, assessing time from HCV diagnosis to treatment start on change in liver function. Analysis was performed using multivariable linear regression models producing risk differences, adjusting for history intravenous drug use (IVDU) and insurance status.

In total, 904 patients were included. On average, patients were 55.0 years old (IQR=10.0), while a majority were Black/African American (70.1%), male (78.2%), insured through Medicaid (60.1%), and did not report a history of IVDU (52.3%). Following HCV diagnosis, hepatis ultrasound occurred a median of 120 days later (IQR=345), Fibroscan occurred a median of 251 days later (IQR=383), fibrosure occurred a median of 232 days later (IQR=507). Among Fibrosure and Fibroscan staging methods, the first fibrosis staging following HCV diagnosis was median of 248 days (IQR= 506), while a median of 216 (IQR=495) days when including hepatic ultrasound. APRI was 0.44 (IQR=0.67) at screening, 0.41 (IQR=0.52) at first fibrosis staging, and 0.36 (IQR=0.44) at treatment. FIB4 was 1.68 (IQR=1.74) at screening, 1.67 (IQR=1.61) at first fibrosis staging, and 1.67 (IQR=1.16) at treatment. For each 180-day delay in fibrosis staging following HCV diagnosis, patients had a 13% (p=0.03) and 20% (p<0.001) increase in hepatic dysfunction, as measured by APRI and FIB4 respectively. For each 180-day delay in initiating treatment following HCV diagnosis, patients had both an 11% increase in hepatic dysfunction by APRI (p=0.28) and FIB4 (p=0.17).

There was a statistically significant relationship on increase in hepatic dysfunction with increasing delay from HCV antibody screening to fibrosis staging. Patients who receive RUQ ultrasound may receive fibrosis staging earlier than those staged by fibroscan or fibrosure tests. Relationships between delays in care on hepatic dysfunction indicates a potential area necessitating further study to better understand whether the requirement for fibrosis staging negatively affects treatment outcomes for patients with HCV.