Liver Cancer Risk Doesn’t End With Successful Hepatitis C Treatment

People who developed hepatocellular carcinoma after being cured of HCV had cirrhosis and worse liver function.

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People with cirrhosis but no existing liver nodules may be at risk for developing hepatocellular carcinoma (HCC), the most common type of liver cancer, even after being cured of hepatitis C, according to study results published in the Journal of Hepatology.

Over time, chronic hepatitis B or C virus (HBV or HCV) infection, fatty liver disease, heavy alcohol use and other causes can lead to cirrhosis, liver cancer and the need for a liver transplant. Direct-acting antiviral therapy can now cure hepatitis C virus in most people, but it does not always reverse existing damage. Those who have attained a sustained virological response (SVR) after treatment can still develop liver cancer, and guidelines recommend that those with cirrhosis should undergo regular monitoring.

It is known that people who already have liver nodules before receiving antiviral therapy for hepatitis C have a higher risk for HCC. But the risk of liver cancer in those without pre-existing liver nodules after achieving a functional cure is unclear.

Maria Reig, MD, PhD, of Hospital Clinic Barcelona and colleagues sought to determine the prevalence of hepatocellular carcinoma among people with severe fibrosis (Stage F3) or cirrhosis (Stage F4) who had been successfully treated for HCV.

The study included individuals who had undergone ultrasound scans that did not find any evidence of liver cancer or other uncharacterized liver nodules within 30 days of achieving SVR.

A total of 185 people, of whom 63 had severe fibrosis and 122 had cirrhosis, were ultimately included. Most people with cirrhosis fell into Child-Pugh Class A (92%), an indicator of well-preserved liver function. Some 43% of people with cirrhosis had portal hypertension, or elevated pressure in liver blood vessels.

The researchers tracked all participants every six months until they developed primary liver cancer, passed away or withdrew from the trial. The median duration of clinical and radiologic follow-up was 52 and 48 months, respectively.

Over the course of the study, 10 participants developed hepatocellular carcinoma. The median time between attaining SVR and developing HCC was 28 months. In addition to HCC, participants also developed 12 secondary liver cancers, or cancers that had spread from other parts of the body.

The prevalence of HCC was 1.46 per 100 person-years across the entire study population. Among people with cirrhosis, the HCC prevalence was 2.24 per 100 person-years, and for those with clinically significant portal hypertension, it rose to 3.63 per 100 person-years. However, none of the participants with severe fibrosis—but not cirrhosis—developed HCC.

“Patients with HCV-related cirrhosis, without non-characterized liver nodules at sustained virologic response, remain at risk of hepatocellular carcinoma despite viral cure,” wrote the researchers. “However, the cancer risk after successful direct-acting antiviral treatment is marginal in patients with F3 fibrosis without non-characterized liver nodules. If confirmed in larger prospective studies, current screening recommendations may need to be revisited in this group of patients.”

Click here to read the study abstract in the Journal of Hepatology.
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