People With HIV and Hepatitis B Need Ongoing Liver Cancer Monitoring

People living with both viruses remain at risk for hepatocellular carcinoma despite antiviral therapy.

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People with HIV and hepatitis B virus coinfection remain at risk for developing hepatocellular carcinoma (HCC) despite antiviral treatment, and they should undergo regular monitoring for liver cancer, according to research presented at the virtual Conference on Retroviruses and Opportunistic Infections (CROI).

Over years or decades, chronic hepatitis B can lead to serious liver problems, including cirrhosis and HCC, the most common type of liver cancer. Some prior research has found that people living with both HIV and hepatitis B virus (HBV) tend to have faster liver disease progression. The American Association for the Study of Liver Diseases recommends that people with cirrhosis should be monitored for HCC using ultrasound imaging and alpha-fetoprotein blood tests every six months.

Nucleoside/nucleotide analogues can keep HBV replication in check indefinitely but they seldom lead to a cure. People with HIV/HBV coinfection are advised to include drugs with dual activity in their antiretroviral regimen. Tenofovir disoproxil fumarate (Viread, also in Truvada and several single-tablet regimens), tenofovir alafenamide (Vemlidy, also in Descovy and single-tablet regimens), lamuvidine (Epivir and combination pills) and emtricitabine (Emtriva and combination pills) are active against both viruses.

H. Nina Kim, MD, of the University of Washington in Seattle, and colleagues evaluated risk factors for liver cancer among people with HIV/HBV coinfection in 22 cohorts that make up the North American AIDS Cohort Collaboration on Research and Design.

Among the nearly 124,000 HIV-positive people included in the cohorts between 1995 and 2016, a total of 9,383 had HIV/HBV coinfection. After excluding those with inadequate data and those who already had HCC, 8,354 people were included in this analysis.

Most participants (93%) were men, 41% were Black and the median age was 43. About a third reported heavy alcohol use, 22% had chronic hepatitis C and 12% had obesity, which are also risk factors for liver disease progression. About three quarters were taking antiretrovirals that are

active against HBV.

The low proportion of women was a limitation of the study, as was the absence of data about hepatitis delta (which sometimes occurs along with HBV and can lead to more aggressive liver disease), fatty liver disease or cirrhosis status.

In the full study population, there were 115 new cases of HCC, or 1.8 per 1,000 person-years. Older age, heavy alcohol use and chronic hepatitis C were independent risk factors for liver cancer. The researchers saw no significant association between HCC and HIV viral load or CD4 cell percentage.

About two thirds of the participants had available quantitative HBV viral load data. In this subgroup, an HBV DNA level above 200 international units per milliliter was associated with a nearly threefold higher risk of developing liver cancer, and the odds were more than fourfold higher for those with HBV levels exceeding 20,000 IU/mL.

Looking at the effect of hepatitis B treatment, sustained HBV suppression for a year or more was associated with a 58% reduction in liver cancer risk, while suppression for four years or more reduced the risk by 66%.

“Our findings underscore that antiviral therapy reduces but does not eliminate the risk of HCC,” Kim said. “The data highlight the importance of HBV surveillance and optimization of HBV suppression. To gain maximal protective benefit from antiviral therapy for HCC prevention, sustained—and ideally uninterrupted—suppression of HBV may be necessary over years.”

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