

Diabetes Tied to Complications and Death Following Hep C Treatment

This finding from a recent study applies to those who do not have cirrhosis when they are treated for the virus.

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Among those who do not have cirrhosis when they are treated for hepatitis C virus (HCV), having diabetes is associated with an increased risk of subsequent liver-related complications and death compared with not having diabetes, according to recent study findings.

Additionally, a high body mass index (BMI) may be linked to a lower risk of death and hepatocellular carcinoma (HCC, the most common form of liver cancer) among those who have been treated for HCV, perhaps because obesity is itself tied to an absence of sarcopenia, or progressive general loss of muscle tissue.

Jihane N. Benhammou, MD, PhD, of the University of California, Los Angeles School of Medicine, presented the study in question at The Liver Meeting, the Annual Meeting of the American Association for the Study of Liver Diseases, in Boston this week.

The study authors sought to determine whether diabetes or obesity are associated with negative liver-related outcomes and death during long-term follow-up after individuals have been treated for HCV with direct-acting antivirals (DAAs).

So they conducted a retrospective cohort analysis of data from the Veterans Affairs Healthcare system on veterans with hep C who were treated with DAAs between 2013 and 2015. The cohort members needed to have follow-up data 12 weeks after completing treatment or beyond.

The study defined obesity as is standard: a BMI of 30 or greater.

Of the 33,003 people included in the study, 30% had diabetes, and 35% were obese.

Of the 9,794 people who had diabetes and the 23,209 who did not, the respective baseline characteristics were as follows: average age, 62 and 61 years old; percentage male, 98% and 97%; percentage white, 42% and 57%; genotype 1 of hep C, 86% and 83%; previous treatment of hep C, 28% and 22%; average BMI, 29.8 and 27.6; cirrhosis before starting DAAs, 40% and 28%; liver cancer before starting DAAs, 3.3% and 2.3%; a MELD score of 9 or greater (indicating an

elevated risk of liver-related mortality), 36% and 27%; and an FIB-4 of at least 3.25 (indicating significant fibrosis, or scarring, of the liver), 40% and 34%.

Of the 11,600 people with obesity and the 21,403 who were not obese, the respective characteristics were as follows: average age, 61 and 61 years old; percentage male, 96% and 97%; percentage white, 52% and 53%; genotype 1 of hep C, 83% and 84%; previous treatment of hep C, 26% and 22%; diabetes, 39% and 24%; cirrhosis before starting DAAs, 35% and 30%; liver cancer before starting DAAs, 2.3% and 2.7%; a MELD score of 9 or greater, 32% and 28%; and an FIB-4 of at least 3.25, 37% and 35%.

During an average three years of follow-up after DAA treatment, 10.1% of the study cohort died, 5.0% developed compensated cirrhosis (the milder form of the severe liver disease, hereafter referred to as simply cirrhosis), 4.7% developed decompensated cirrhosis (the more severe form) and 4.0% were diagnosed with liver cancer.

After adjusting the data to account for various differences between the study cohort members, the investigators found that among those treated with DAAs and cured of HCV, having diabetes, compared with not having diabetes, was associated with a 1.14-fold increased risk of death, a 1.36-fold increased risk of cirrhosis, a 1.19-fold increased risk of decompensated cirrhosis and no difference in the risk of liver cancer.

Among those with cirrhosis when they were treated with DAAs, diabetes was not associated with any difference in the risk of mortality, decompensated cirrhosis or liver cancer. However, among those without cirrhosis, diabetes was associated with a 1.22-fold, 1.34-fold and 1.74-fold increased risk of death, cirrhosis and decompensated cirrhosis, respectively.

Among those cured of hep C, obesity, compared with having a normal weight (a BMI between 18.5 and 24.5), was associated with a 1.28-fold increased risk of cirrhosis and a 23% lower risk of liver cancer.

Among those treated with DAAs but not cured of hep C, obesity was associated with a 1.57-fold increased risk of cirrhosis, a 27% lower risk of death and a 30% lower risk of liver cancer.

The study was limited by the fact that muscle mass and sarcopenia were not captured among the participants. Nor were non-alcoholic fatty liver disease (NAFLD) or non-alcoholic steatohepatitis (NASH) assessed.

The study authors indicated various research avenues to which their findings may lead. These include: assessing the risk of NAFLD in those treated with DAAs, identifying non-invasive markers among those cured of HCV who have obesity or diabetes and who would benefit from screening, assessing the effects of statin use in DAA-treated individuals with a high BMI and determining whether the associations identified in their research persist during longer follow-up.

To read the study abstract, visit

https://plan.core-apps.com/tristar_aasld19/abstract/0b8716de6c519b8c26b6b46a114ef383.

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