Despite ARV Therapy, HIV/Hep C Patients Face Increased Risk of Serious Liver Disease

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Even with the use of antiretroviral (ARV) therapy, U.S. veterans living with both HIV and hepatitis C virus (HCV) face a higher risk of serious liver disease, compared with people only infected with HCV (monoinfection), according to sobering new data reported Wednesday, July 25, at the XIX International AIDS Conference (AIDS 2012) in Washington, DC.

Vincent Lo Re, MD, of the University of Pennsylvania, who presented the research on behalf of the Veterans Aging Cohort Study, began his overview by noting that poor liver function and liver cancer are serious concerns among people coinfected with both HIV and HCV. Though studies have confirmed that ARV therapy can slow the progression of liver scarring (fibrosis) in coinfected individuals, there has been very little research conducted to see if ARV-treated coinfected patients are any less likely to experience poor liver function and liver disease-related complications compared with HCV-monoinfected patients.

To explore this, Lo Re and his colleagues compared the incidence of hepatic decompensation—a marked decline in liver function, often accompanied by symptoms of illness—between ARV-treated coinfected and HCV-monoinfected veterans.

To do this, they reviewed electronic medical record data for 4,280 coinfected patients and 6,079 HCV-monoinfected patients. All patients included in the analysis had detectable HCV viral loads and at least 12 months of follow-up information available; none of the patients had ever been treated with an interferon-based regimen for their hepatitis.

All HIV/HCV-coinfected patients were required to be receiving ARV therapy.

At the start of the follow-up period, the average age of the cohort participants was 48 years; approximately 99 percent were male, 63 percent were black, 28 percent had a history of alcohol dependence and the majority had high HCV viral loads (400,000 or higher). The average pre-ARV therapy HIV viral load in the coinfected patients was 100,000; 45 percent had a CD4 count at or below 200 cells. The average length of follow up included in the analysis was 6.8 years among the

coinfected patients and 9.9 years among the HCV-monoinfected patients.

Lo Re and his colleagues found significantly higher rates of hepatic decompensation among the coinfected patients during the follow-up period, compared with the monoinfected patients (6.3 versus 5 percent, respectively).

Statistically, this calculated into 83 percent greater likelihood of hepatic decompensation occurring among all patients in the study living with both HIV and HCV. When the researchers focused only on those who had very low viral loads—below 400 copies—the risk of hepatic decompensation was still increased by 73 percent.

The average age at the time hepatitis decompensation, noted in the patients' medical records, was similar in both groups: 52 among the coinfected patients and 53 among the HCV-monoifected patients.

One of the hallmark complications of hepatic decompensation—bleeding in the esophagus or stomach (variceal bleeds)—was significantly more likely to be documented among coinfected patients (55 verus 26 percent).Rates of other common complications of decomensation, notably a buildup of fluid in the abdomen (ascites) or abdominal bacterial infections (peritonitis), were similar between the two groups.

Rates of liver cancer were roughly similar between the two groups (1.2 verus 0.9 percent). However, Lo Re's group determined that the risk of liver cancer was still 69 percent higher among the HIV/HCV-coinfected patients, compared with those living with HCV alone.

Death rates were substantially higher among the coinfected patients, compared with the monoinfected patients (32.9 versus 15.4 percent). However, Lo Re and his team noted that liver-related deaths were significantly more likely to occur among HCV-monoinfected patients (20.1 versus 7.8 percent).

While these data are sobering, Lo Re noted a handful important limitations of his team's study. For one, the researchers were unable to accurately determine how long patients in either group had been living with HCV. And with limited liver biopsy data, it wasn't clear if patients in one group were more or less likely to have advanced liver fibrosis—a precursor of hepatic decompensation—at the start of the follow-up period, compared with those in the other group. Finally, as these results involved military personnel, they may not be directly applicable to general populations living with HCV monoinfection or HIV/HCV coinfection.

Nevertheless, Lo Re concluded that "despite antiretroviral therapy, HIV/HCV coinfected patients had higher risk of hepatic decompensation than HCV-monoinfected patients. " As for future research directions, he added it will be important to further evaluate the risk factors associated with the increased risk of hepatic decompensation among coinfected patients otherwise responding well to HIV treatment and to developed a clear and concise monitoring tool—a predictive index—to help clinicians manage these risk factors before severe liver disease occurs.

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