

Hepatitis C

HBV/HCV Coinfection

Due to overlapping transmission routes, many people are infected with both hepatitis B and C. In a study published in the November 2009 *Liver International*, J.W. Yu and colleagues evaluated virological characteristics of 50 Chinese patients coinfecting with HBV and HCV, as well as the efficacy of pegylated interferon alfa-2a (Pegasys) plus ribavirin. Most coinfecting patients (92%) had HCV as their dominant virus type; their average HBV DNA level was lower than usually seen in HBV mono-infected people and they were significantly less likely to be hepatitis B "e" antigen positive (12% vs. 45%). Several prior studies have shown that HBV and HCV suppress each others' replication.

HBV/HCV coinfecting patients with HCV genotype 1 had significantly higher early response rates than HCV mono-infected individuals (50% vs. 16% partial early virological response at week 12; 90% vs. 56% end-of-treatment response). However, the coinfecting group also had a higher relapse rate (56% vs. 21%), and therefore ended up with a sustained virological response (SVR) rate similar to that of HCV mono-infected patients (40% vs. 44%). Among patients with HCV genotype 2, response rates were similar at all time points. While coinfecting patients typically had suppressed HBV, they risked rising HBV levels after HCV was cured. Those who achieved SVR were nearly four times more likely than nonresponders to experience HBV reactivation (33% vs. 9%).

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Who Gets HCV Treatment?

Some people who most need treatment for chronic hepatitis C are not receiving it, according to two recent journal articles. As reported in the December *Hepatology*, M. Volk and colleagues looked at the public health impact of hepatitis C treatment in the U.S. Using an electronic audit of pharmacies nationwide, they estimated that approximately 663,000 patients received a new prescription for pegylated interferon between 2002 and 2007. The rate of treatment appeared to decline over time, with fewer prescriptions written in later years (falling from 126,000 in 2002 to 83,000 by 2007). Based on a mathematical model, they projected that if this trend continues, only 14.5% of liver-related deaths due to HCV during 2002-2030 would be prevented by antiviral therapy, and that fewer than 1.4 million people with HCV (out of an estimated 3.9 million total) would be treated by 2030.

Using data from the National Health and Nutrition Evaluation Survey (NHANES) Hepatitis C Follow-Up Questionnaire, they found that the primary barrier to treatment is lack of diagnosis, as only 49% of the 133 respondents were previously aware they were infected. Among people with HCV,

24% did not have treatment recommended by their physician, 9% did not follow up with their provider, and 8% refused treatment, leaving 12% who received therapy. Barriers to treatment included lack of health insurance, limited access to medical care, and the low rate of HCV screening by primary care doctors, the researchers suggested. "Efforts to improve rates of diagnosis and treatment will be required if the future public health burden of hepatitis C is to be ameliorated," they concluded.

In a related study published in the November 2009 *Journal of Health Care for the Poor and Underserved*, D. Alfandre and colleagues evaluated clinical and socio-demographic characteristics associated with failure to start hepatitis C treatment. This retrospective study focused on a multiethnic cohort of treatment-naïve patients seen at a primary care hepatitis C clinic in New York City between January 2003 and May 2007.

Out of 168 eligible patients, 24% started treatment. According to a multivariate analysis, individuals with HCV genotypes 1 and 4 were less likely than those with genotypes 2 or 3 to initiate therapy (21% vs. 42%, respectively). Unmarried people and those with more medical comorbidities were also less

likely to start treatment. However, age, sex, race/ethnicity, and language did not affect the likelihood of treatment.

Does Coffee Slow Fibrosis?

Research continues to accumulate showing that coffee may have a beneficial effect on liver health. As reported in the November 2009 *Hepatology*, N. Freeman from the National Cancer Institute and colleagues assessed the relationship between coffee consumption and liver disease progression among 766 chronic hepatitis C patients with bridging fibrosis or cirrhosis enrolled in the HALT-C trial who did not achieve sustained response to pegylated interferon plus ribavirin.

At study entry, participants who drank more coffee had significantly less severe steatosis, lower AST-to-ALT ratio, lower alpha-fetoprotein (a marker for liver cancer), less insulin resistance, and higher albumin levels than non-coffee-drinkers. Over the nearly four-year follow-up period, participants who drank three or more cups of coffee per day had a 53% lower risk than non-coffee-drinkers of clinical outcomes including ascites, hepatic encephalopathy, bacterial peritonitis, bleeding varices, hepatocellular carcinoma, in-

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creased fibrosis, and liver-related death. Outcome rates decreased from 11.1 per 100 person-years for patients who drank no coffee, to 8.2 per 100 person-years for those who drank 1-2 cups per day, to 6.3 per 100 person-years for those who drank three or more cups per day. The researchers did not see a similar effect among individuals who drank black or green tea. "In a large prospective study of participants with advanced hepatitis C-related liver disease," they concluded, "regular coffee consumption was associated with lower rates of disease progression."

HIV/HCV Coinfection and Fibrosis

Prior research indicates that HIV positive people with chronic hepatitis C tend to experience more rapid liver disease progression than HCV monoinfected individuals. In a study described in the November 2009 *Journal of Viral Hepatitis*, L. Martin-Carbonero and colleagues looked at liver fibrosis progression among chronic hepatitis C patients with persistently normal ALT. The study included 449 HIV negative and 133 HIV/HCV coinfecting patients not previously treated for hepatitis C who underwent liver fibrosis assessment since 2004 at three European hospitals using the noninvasive transient

elastometry (FibroScan) method. Most of the HIV patients were on combination antiretroviral therapy and had a high average CD4 cell count of 525.

HCV Does Not Speed HIV Progression

While a considerable body of data indicates that HIV accelerates hepatitis C liver disease progression, evidence is conflicting about the effect of HCV on HIV disease progression.

As reported in the November 15, 2009, *Clinical Infectious Diseases*, T.Y. Chen and colleagues performed a meta-analysis to estimate the effect of coinfection on HIV disease progression and mortality before and after the advent of highly active combination antiretroviral therapy (HAART) in 1996. Searching the PubMed and EMBASE databases through April 2008, they identified 10 relevant studies from the pre-HAART era with about 14,600 total participants, and 20 studies from the HAART era with about 87,000 total participants.

In the pre-HAART era, HIV/HCV coinfecting individuals had a slightly lower risk of death due to any cause than HIV monoinfected people (risk ratio 0.68). In the HAART era, however, coinfecting patients had a 35%

higher overall mortality rate (risk ratio 1.35). The risk of AIDS diagnosis did not differ significantly between the two groups, and the risk of death increased with longer duration of HIV/HCV coinfection. The researchers concluded that, "HCV coinfection did not increase mortality among patients with HIV infection before the introduction of HAART. In contrast, in the HAART era, HCV coinfection, compared with HIV infection alone, increases the risk of mortality, but not the risk of AIDS-defining events." While HIV positive people in the pre-HAART era typically did not survive long enough to develop advanced HCV-related liver damage, liver disease has now become a major cause of death for coinfecting individuals. They recommended that, "Future studies should determine whether successful treatment of HCV infection could reduce this excess risk of mortality in coinfecting patients."

