Researchers disprove long-standing belief about HIV treatment

July 25 2008

Researchers at Wake Forest University Baptist Medical Center have disproved a long-standing clinical belief that the hepatitis C virus slows or stunts the immune system's ability to restore itself after HIV patients are treated with a combination of drugs known as the "cocktail."

Hepatitis C (HCV) infection is more serious in HIV-infected people, leading to rapid liver damage, according to the Centers for Disease Control. Intravenous drug use is a main method of contraction for both HIV and HCV and 50 to 90 percent of HIV-infected drug users are also infected with HCV.

The Wake Forest Baptist study looked at whether having HCV co-infection impairs immune restoration in patients receiving highly active anti-retroviral therapy (HAART) to suppress their HIV infection. The results appear in the July issue of *AIDS Research and Human Retroviruses*.

The research focused on levels of CD4 cells, the specific type of immune cell that is attacked by the HIV virus, and their ability to rebuild after HIV is suppressed.

"We've been observing that in some patients that are co-infected with hepatitis C, we were treating their HIV with HAART but didn't always get very good restoration of CD4," said Marina Nunez, M.D., lead researcher and an assistant professor of infectious diseases. "Some studies suggested it was because of the hepatitis C. This study says it's

not the presence of active hepatitis C replication."

Thus, said Nunez, genetic factors involved in the immune system regulation, confounding factors associated with HCV acquisition, or other unknown factors might explain the blunted immune restoration observed in some co-infected patients. "Research efforts should pursue the role of those other factors in the immune restoration," she said.

"From a clinical standpoint, although these findings will not alter the clinical management of HIV-HCV-co-infected patients, they make clear that even after successful treatment of the HCV infection, some patients may still not get an adequate CD4 recovery under HIV treatment."

For the retrospective study, researchers examined existing medical records of 322 patients from two separate databases – one from Madrid, Spain, and the other from Wake Forest University Baptist Medical Center. Patients were separated into two groups – those co-infected with hepatitis C and HIV and those infected only with HIV. Researchers reviewed CD4 levels at baseline (before beginning HIV suppression) and every year after for up to three years, while the patients continuously received HAART, an HIV treatment consisting of three different types of medicines used by many patients, and formerly referred to as the HIV "cocktail."

Years of clinical experience have shown that, with HAART treatment suppressing the HIV, CD4 levels are typically able to restore themselves, Nunez said.

However, in some patients, it has been observed that the immune restoration is poorer after HAART. Therefore, Nunez said, it has been a common practice for doctors to attribute less than desirable CD4 restoration after HAART in co-infected patients to the hepatitis C virus.

Studies to date have found evidence both in support of and against this belief, Nunez said. But a limitation in previous studies has been that co-infected patients have been identified by the presence of HIV and the hepatitis C antibody. Since many patients with hepatitis C clear the active virus but continue to carry the antibody, there hasn't been a pure sample of patients truly co-infected with both active viruses to analyze. In this study, only patients with HIV and active hepatitis C cell replication, therefore active virus, were classified as "co-infected."

The study found that there is no difference in the CD4 restoration of co-infected patients and mono-infected patients. However, it did show some differences that seem to be associated with age, gender or past intravenous drug use.

"The purpose of this study was to find out if hepatitis C was impeding the CD4 restoration in co-infected patients," Nunez said. "And it does not. There are other factors doing it. This study says that you can look into those other factors, but we cannot blame the hepatitis C anymore."

Source: Wake Forest University