Statins Show Promise as Anti-inflammatory Therapies for HIV Infection

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Beyond their ability to lower cholesterol, a class of drugs called statins may hold a key health benefit for people living with HIV, as demonstrated in a recent study to be published in The Journal of Infectious Diseases evaluating the effects of the drug Lipitor (atorvastatin) on potentially harmful markers of immune activation and inflammation.

Statins are well known for their effects on lipids and are commonly prescribed for people unable to control their cholesterol levels through diet and exercise alone. Studies confirm that statins reduce the risk of cardiovascular disease (CVD), but researchers have long suggested that the lower incidence of CVD among those using the drugs can't be explained by their lipid-lowering potential alone.

Additional studies have shown that statins can lower blood levels of an inflammatory protein called C-reactive protein, which has been independently linked to CVD risk. There's also data indicating statins can tone down certain aspects of immune activation, such as CD4 and CD8 cell proliferation, which may also play a role in CVD.

A clinical trial of the statin Mevacor (lovastatin), reported in 2005, suggested that these drugs may also reduce HIV replication. Three other studies conducted since—testing Lipitor, Zocor (simvastatin) or Pravachol (pravastatin)—failed to duplicate these findings.

The effects of statins on inflammation and immune activation may bode well for people living with HIV, given that both inflammation and immune activation are frequently noted in the presence of chronic viral infections and have been associated with more rapid disease progression and an increased risk of various non-AIDS-related health complications, such as CVD.

In turn, Anuradha Ganesan, MD, of the National Naval Medical Center and her colleagues set out to explore the potential positive effects of one statin in particular, Lipitor, in a small number of people living with HIV.

The clinical trial involved 24 patients who were not on antiretroviral (ARV) therapy and who had blood levels of "bad" LDL cholesterol that qualify for statin treatment; the study randomized them to receive either eight weeks of Lipitor (80 mg) or placebo daily. After a four-to-six-week washout period, in which nobody received treatment, those originally randomized to Lipitor received the placebo (and those originally randomized to the placebo took Lipitor) for another eight weeks.

Twenty-two of the 24 randomized patients completed the study.

The study's primary goal was to determine whether Lipitor had any effect on viral load in the patients, hence one of the reasons why patients were not permitted to use proven ARVs in the clinical trial. No affects on HIV replication were documented during the eight weeks of Lipitor treatment, compared with those who received placebo.

The authors note, however, that statins may have an additive effect on viral replication when combined with (ARV) therapy. "Given the recognized benefits of [ARV therapy] and the improved access to [ARVs], it is likely that in the future the majority of patients will receive [ARV therapy]. Thus, studies designed to evaluate the adjunctive effects of statins in [ARV]-treated individuals with viral suppression are likely to have clinical relevance."

Statistically significant reductions in some markers of inflammation and immune activation, such as HLA-DR and CD38, were reported in those received Lipitor, compared with those receiving the placebo. These results mirrored those of a different study that employed low-dose Lipitor over a 14-day period in HIV-negative volunteers. Whether or not these reductions in inflammation and immune activation markers are clinically significant—meaning associated with slower disease progression or a reduced risk of health complications—has not yet been determined.

The reduced inflammation and immune activation did not correlate with changes in LDL levels in the study, confirming that statin activity is multifaceted and may have several advantages in people living with HIV.

An accompanying editorial, authored by Andrew Carr, MD, of St. Vincent's Hospital in Sydney, sums up these and similar data generated over the past several years. "Unless other statins have modes of action that are different from those of high-dose atorvastatin," he writes, "it seems unlikely that other statins will be found to suppress HIV replication. However, the present data suggest that statins merit evaluation over longer periods in HIV-infected adults who are receiving effecting antiretroviral therapy but who have persistent T cell activation, given that ongoing inflammation in HIV-infected adults receiving therapy is associated with a greater risk of HIV disease progression and death."

Car concludes: "A very large study would probably be required to determine whether the potentially positive effects of statin therapy in inflammatory markers will translate into less HIV disease progression and fewer cases of inflammatory non-AIDS-related illnesses, such as cardiovascular disease and end-stage liver disease."

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