Inflammatory Cells Linked to Earlier Death From AIDS, Hep C

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Certain inflammatory markers are linked to an earlier death among alcohol abusers with HIV, including those coinfected with hepatitis C virus (HCV). This possibly helps explain why, despite antiretroviral (ARV) treatment, some HIV-positive people die before others. Publishing their findings in the journal AIDS, researchers followed 400 HIV-positive abusers of alcohol, half of whom also had hepatitis C, for three to five years between 2001 and 2009. The participants were tested for seven pro-inflammatory cytokines upon their entry into the study and were tracked through national databases to verify whether or not they had died by the study's end.

Cytokines are proteins that alert white blood cells to flock to a zone of inflammation. In the short term, this process is good for the body; it is, for example, how the a bacterial or fungal infection is cleared. But in the long-term it is harmful. HIV causes chronic inflammation, and much current research in the field focuses both on how the inflammation harms the body and on ways to fight the inflammation.

Eighty-five of the participants eventually died, mostly of AIDS- or hepatitis C-related causes. The researchers found that higher indicators of inflammation were strongly linked to increased risk of death in these heavy drinkers. The connection remained regardless of whether or not an individual was taking ARVs. The inflammatory marker known as interleukin-6 (IL-6) had the most significant correlation to mortality.

"Current antiretroviral drug regimens may be able to improve mortality in most patients, but are unable to decrease the potentially dangerous burden of a chronic inflammatory state in the body," Daniel Fuster, MD, PhD, a researcher at the Clinical Addiction Research and Education (CARE) unit at Boston University School of Medicine, and the study's lead author, said in a release. "Additional research should explore how to better manage chronic inflammation in these patients."

To read the study release, click here.