Viral Reactivation a Likely Link between Stress and Heart Disease

A new study could provide the link that scientists have been looking for to confirm that reactivation of a latent herpes virus is a cause of some heart problems. Looking at blood samples from 299 heart patients, researchers at Ohio State University found that those who had suffered a heart attack were the most likely to have inflammatory proteins circulating in their blood compared to patients with less acute symptoms. And having more of one of these proteins in the blood was linked to the presence of antibodies that signal a latent Epstein-Barr virus (EBV) reactivation.

To date, these relationships have been hard to find because scientists have been unable to detect evidence of a virus in diseased areas of the cardiovascular system.

In this study, however, the researchers instead looked for antibodies against a protein that can be produced even when only partial or incomplete reactivation of Epstein-Barr EBV occurs. And when this antibody was detected, it was associated with immune system malfunctions connected to inflammation—a known risk factor for heart disease.

Identifying a solid link between a reactivated virus and heart disease is important because of the prevalence of EBV, a human herpes virus that causes infectious mononucleosis and several different types of tumors. An estimated 95 percent of Americans have been infected with the virus by adulthood, and once a person is infected, the virus remains dormant in the body. It can be reactivated without causing symptoms of illness, but reactivation has potential to create chaos in the immune system.

Stress is a known predictor of reactivation of EBV, meaning virus reactivation could be a mechanism by which stress leads to chronic inflammation and eventually cardiovascular diseases.

“In the big picture, this may help clarify the role these viruses play in heart disease,” said co-author Ron Glaser, director of Ohio State’s Institute for Behavioral Medicine Institute (IBMR) and professor of molecular virology, immunology and medical genetics. “And it makes sense, because we know that some viral proteins can induce inflammation, affecting the lining of blood vessels, so that inflammation is in the right place to function as a significant risk factor for heart disease.”

The research is published in the online journal *PLOS ONE*.

The patients whose blood was sampled for the study were undergoing angioplasty to clear narrow arteries. Researchers tested their blood for the presence of

numerous cytokines – proteins that signal the presence of inflammation – as well as for antibody to an EBV encoded viral protein called dUTPase. This protein is produced early in the process of viral reactivation, and may be present even if signs of the virus itself cannot be detected.

Co-author Marshall Williams, professor of molecular virology, immunology and medical genetics, uses a highly sensitive method to detect these antibodies, and hopes to develop an equally effective technique that could be put to use in clinical laboratories.

Patients who had had acute myocardial infarction – a heart attack – were the most likely to have the highest measures of two cytokines, interleukin-6 (IL-6) and intercellular adhesion molecule 1 (ICAM-1) in their blood compared to patients whose main symptom was chest pain.

Researchers also identified a strong relationship between circulating concentrations of ICAM-1 and detectable antibodies to EBV dUTPase. In fact, the highest values of ICAM-1 were found in patients who had had a heart attack and were positive for the dUTPase protein. A similar trend was seen with IL-6, but the finding could have been attributed to chance.

“This study provides the essential clinical corroboration of this mechanism showing enhanced levels of proinflammatory proteins in the blood of patients with acute coronary events and detectable levels of the EBV-related protein,” said Philip Binkley, professor of cardiovascular medicine and epidemiology at Ohio State and a lead author of the study.

Source: Ohio State University [1]

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