CMV, HIV and Immune Activation

August 5, 2010 By Joseph Sonnabend, MD

Always on the lookout for information on cytomegalovirus (CMV) an article entitled “Human Cytomegalovirus Induces Systemic Immune Activation Characterized by a Type I Cytokine Signature” caught my eye this morning prompting yet another post, this time a short one on the role of CMV in HIV disease.

In a previous post I wrote about the contribution of CMV to the immune activation and associated chronic inflammation that probably is at the heart of the pathogenesis of HIV disease.

In this article, a team in the Netherlands reports that CMV infection results in persistent immune activation associated with a systemic inflammatory response. CMV like all herpes viruses becomes latent after an acute infection and can be periodically reactivated. Unlike other herpes viruses the immune activation and inflammation associated with acute CMV infection persists even when the virus becomes latent, at least as far as this has been studied. The article reports that among other abnormalities, C reactive protein (CRP) levels and gamma interferon levels are elevated in primary CMV infection persisting into latency.

The authors note that “CMV infection appears to induce a chronic proinflammatory state not only in immunosuppressed patients but also in healthy individuals”. They then go on to speculate that CMV infection may contribute to cardiovascular disease by promoting atherosclerosis, and also to immunosenescence, the aging of the immune system.

Immune activation, chronic inflammation, atherosclerosis, immunosenescence, elevated CRP levels, and atherosclerosis. We have become familiar with all these terms from descriptions of HIV disease.

None of these patients were HIV infected.

But virtually all HIV infected individuals are also infected with CMV.

So these results are relevant to HIV disease. They provide further support for a view that CMV is a major contributor to the immune activation characteristic of HIV disease as described in previous posts. Both viruses contribute to immune activation and reciprocally enhance the replication of each other in a positive feedback relationship.

In 1981, the year the first cases of AIDS were reported, some of us then believed that CMV played

a major role in the disease. In 1983 I published a theoretical model that described a process involving repeated infections with CMV that could result in disease. This was at a time before HIV had been discovered, and all we had were theories. This partly explains why 30 years later, I'm so interested in the renewed attention given to this virus.

The theory that reinfection with CMV plays a role in AIDS was the basis for the first recommendations for the use of condoms to prevent the spread of AIDS, made in a booklet written by Michael Callen and Richard Berkowitz. Now, thanks to Richard, the booklet, "How to have sex in an epidemic: one approach" can be be seen 27 years after it was published.

How this booklet came to be written before the discovery of HIV is an interesting story which I'll describe in another post.

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