HIV and Herpes Viruses

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I'm returning to this topic yet again as another paper has appeared reporting that treatment of HIV infected individuals with Valtrex lowered HIV viral loads to a greater extent than acyclovir. As has become usual in these reports the authors only studied individuals who were infected with herpes simplex virus type 2, which therefore suggests that the effect of Valtrex results from a suppression of this virus.

This is perplexing. Although prevalence of herpes simplex virus type 2 is 50%-90% in some African countries, prevalence of HSV-1 must be even higher. There are several reports that indicate that herpes simplex virus type 1 can activate HIV (as do other herpes viruses), It is just as, and may even be more, sensitive to acyclovir. EBV is also sensitive to this drug, and while CMV is resistant, the high dose of Valtrex may have had some effect on this virus.

There is considerable evidence that CMV plays an important role in the pathogenesis of HIV disease. It's likely that EBV also does. I have written about this in several previous posts, two can be seen here and here.

These trials of the effects of anti-herpes treatment on HIV also represent missed research opportunities. Any effect of treatment on EBV could have been investigated because this would likely be reflected in changes in EBV antibody reactivation patterns. Although CMV is resistant to acyclovir, there may have been an effect of the dose of Valtrex used, which might have been detectable by virus isolation or quantitative PCR. If sera and stored blood samples exist this still may be possible.

Immune activation is at the heart of the pathogenesis of HIV disease. By now it's recognized that factors in addition to HIV contribute to the sustained immune activation that's characteristic of HIV disease. There is much evidence to implicate herpesvirus infections as one of these factors; in addition these viruses can interact with HIV in several other ways, to facilitate infection as well as replication.

The lowering of HIV viral loads by treatment of herpesvirus infections also has implications for

transmission and therefore for prevention. Transmission of HIV is enhanced when viral loads are high.

There is a relationship between viral load and infectivity, although this has been studied in relation to sexual transmission, there can be little question that it also applies to blood borne infection. This is a much neglected source of HIV transmission in regions with generalized epidemics particularly Africa, which can occur in unsafe medical facilities and from other skin piercing procedures with reused and contaminated equipment including the reuse of syringes. This is well described in a recent blog on this site by Simon Collery and David Gisselquist.

It's not only herpesvirus infections that interact with HIV to increase its replication. A number of endemic and waterborne infections in Africa can also do so. I have also written about this in this POZ blog. And more extensively, here.

Treating and preventing these endemic and waterborne infections is an absolutely appropriate part of the attempt to control the HIV epidemic. Of course doing so is important in its own right and will improve the lives of people. These infections shorten life and are debilitating, with destructive social and economic consequences.

Treating many of these infections can lower HIV viral loads to a greater or lesser extent, also noted in this blog.

Should we not also extend the concept of treatment as prevention to the treatment and prevention of endemic and waterborne infections?

By reducing immune activation, and thus HIV replication, this will not only lower HIV viral loads and slow disease progression, but will also improve the lives of millions of people.
