



Zoster is more than ‘just’ a viral infection

“It’s probably just some kind of virus.” How often have we passed that message on to our patients, as buzz words for an illness that we don’t fully understand, can’t give a name to, and expect to be self-limited? We sometimes use “virus” as a symbolic vector, much as our predecessors evoked miasmas. In the acute setting, we are often correct that the illness is self-limited.

The human immunodeficiency virus and hepatitis C epidemics have taught us strong lessons about the varied biological impact of chronic viral infection. But we have much still to learn about the interplay of host-viral interactions. The herpesviruses in particular are not just transient bad humors of the night. Nagel and Gilden remind us in this issue (page 489) that for some viruses—in this case, varicella-zoster virus—striking, and confusing, clinical manifestations can appear long after the initial infection.

Some viruses perform a high-wire balancing act in concert with the host’s immune system. Chickenpox early in life may reappear as shingles later in life, with the virus emerging from its neuronal haven to cause dermatomal or blood-borne systemic zoster. The waning of antiviral immunity has been offered as one explanation for this series of events, and a zoster vaccine is available for healthy adults older than 60 years. However, this is a live vaccine, and it is not generally given to immunosuppressed patients, ie, those who may be at greatest risk for complications of unchecked viral replication.

The nonzoster delayed complications of herpes infection are myriad. Many manifest as neurologic or vascular syndromes, as highlighted in the current article. Other herpesviruses are associated with chronic meningitis, Kaposi sarcoma, and lymphoproliferative diseases. In a beautiful series of experiments, Herb Virgin and colleagues at Washington University showed that mice unable to immunologically cope with a usually benign herpes virus develop and die from an arteritis similar to Takayasu arteritis (at present an idiopathic syndrome in humans).

But latent herpes viral infections may not be all bad: Dr. Virgin’s group also has data that latent herpes infections can prime the innate immune system to more effectively combat certain bacterial infections.

Thus, the patient may have a viral infection, but it may not be “just” a viral infection.

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