Varicella Zoster Virus infection: Bad in adults

Varicella zoster virus (VZV), is a human herpesviruses. The primary infection causes varicella (chicken pox), after which the virus becomes latent in cranial nerve ganglia, dorsal root ganglia, and autonomic ganglia along the entire neuraxis. As cell-mediated immunity declines in elderly individuals and other immunocompromised populations such as people with AIDS, VZV reactivates from 1 or more ganglia to cause herpes zoster (shingles). Although pain, known as postherpetic neuralgia is a frequent complication, a debilitating and life-threatening complication of zoster also occurs and is known as VZV vasculopathy, a cause of transient ischemic attack (TIA) and stroke. Studies of the pathogenesis of disease reveal that upon reactivation from ganglia, VZV travels transaxonally to the adventitia of arteries where productive infection is established, followed by transmural migration of virus to the arterial media and intima. Inflammatory cells, CD4+ and CD8+ T cells, CD68+ macrophages, CD20+ B cells and myofibroblasts are deposited throughout the arterial adventitia and intima, (not the media which is an immuneprivileged site), causing thickening, inflammation and remodeling of the vessel wall, increasing the risk of stroke. Studies have shown that treatment with antiviral therapy following reactivation of VZV decreases the risk of stroke. However not all patients with VZV reactivation and resulting vasculopathy present with the cutaneous lesions, making it difficult to identify and administer therapy.

Nagel, M. et al. 2014. Editorial Commentary: Varicella Zoster Virus Infection: Generally Benign in Kids, Bad in Grown-ups. *Oxford Journals*.