Varicella zoster virus

Varicella zoster virus or varicella-zoster virus (VZV) is one of Herpesviridae known to infect humans. It causes chickenpox (varicella), a disease most commonly affecting children, teens, and young adults, and herpes zoster (shingles) in older adults; shingles is rare in children. VZV is a worldwide pathogen known by many names: chickenpox virus, varicella virus, zoster virus, and human herpesvirus type 3 (HHV-3). VZV infections are species-specific to humans, but can survive in external environments for a few hours, maybe a day or two.

VZV multiplies in the lungs, and causes a wide variety of symptoms. After the primary infection (chickenpox), the virus goes dormant in the nerves, including the cranial nerve ganglia, dorsal root ganglia, and autonomic ganglia. Many years after the patient has recovered from chickenpox, VZV can reactivate to cause neurologic conditions.

Following viral reactivation many years later VZV causes herpes zoster (shingles) as well as a variety of other neurological syndromes. The molecular mechanisms of the conversion of the virus from a lytic to a latent state in ganglia are not well understood. In order to gain insights into the neuron-virus interaction, we studied virus-induced apoptosis in cultures of both highly pure terminally differentiated human neurons and human fetal lung fibroblasts (HFL). It was found that (a) VZV DNA did not accumulate in infected human neurons; (b) VZV transcripts were present at lower levels at all days studied post-infection in neurons; © Western blot analysis showed less VZV IE 63 and very little detectable VZV gE proteins in infected neurons compared with HFL; (d) lower levels of the apoptotic marker cleaved Caspase-3 protein were detected in VZV-infected neurons compared with HFL, and higher levels of the known anti-apoptotic proteins Bcl2, Bcl-XL and also the mitochondrial MT-CO2 protein were found in VZV-infected neurons compared with uninfected cells; and (e) both the MT-CO2 protein and VZV IE 63-encoded protein were detected in infected neurons by dual immunofluorescence. These findings showed that neurons are resistant to VZV-induced apoptosis, which may have relevance to the switching of VZV from a lytic to latent ganglionic neuronal infection [1)].

Diagnosis

The most sensitive method for confirming a diagnosis of varicella is the use of polymerase chain reaction (PCR) to detect VZV in skin lesions (vesicles, scabs, maculopapular lesions). Vesicular lesions or scabs, if present, are the best for sampling.

Prevention

see Varicella vaccination

Treatment

Adults with herpes zoster can be treated with oral acyclovir at a dose of 800 mg five times daily. The recommended dose of intravenous acyclovir for VZV infections is 10 mg/kg every 8 hours, although higher doses (12–15 mg/kg) are sometimes used for life-threatening infections, especially in immunocompromised patients.

Complications

Findings suggested that Delayed facial palsy after microvascular decompression for hemifacial spasm was caused by a re-activation of varicella zoster virus [2].

1)
Kennedy PG, Graner MW, Gunaydin D, Bowlin J, Pointon T, Yu X. Varicella-Zoster Virus infected human neurons are resistant to apoptosis. J Neurovirol. 2020 Mar 3. doi: 10.1007/s13365-020-00831-6. [Epub ahead of print] PubMed PMID: 32125664.
2)
Furukawa K, Sakoh M, Kumon Y, Teraoka M, Ohta S, Ohue S, Hatoh N, Ohnishi T. [Delayed facial palsy after microvascular decompression for hemifacial spasm due to reactivation of varicella-zoster virus]. No Shinkei Geka. 2003 Aug;31(8):899-902. Japanese. PMID: 12968493.