Varicella Zoster Vaccines: Their Implications and Potential Complications

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In early 1970s, a live attenuated varicella vaccine, the Oka strain, was developed in Japan by Takahashi, et al. In 2005, a live attenuated vaccine for herpes zoster was developed by Oxman and his colleagues. Although the varicella vaccine was developed in Japan, the largest experience with this vaccine comes from the United States. Due to the absence of suitable animal models for varicella disease, the earliest tests of vaccine efficacy were carried out directly in humans. Gershon, et al. in 1984, White in 1997, and Sharrar, et al. in 2000, demonstrated in both pre- and post-licensure studies that the vaccine was extremely safe. The vaccine may be administered either by intramuscular or subcutaneous injection. About 20% - 25% of the healthy individuals demonstrate a sore arm after vaccine injection, whereas about 5% of them develop minor rash resembling mild varicella, that usually appear one month after immunization. Mild fever is experienced in about 15% of them. Varicella vaccine should be administered as soon as possible, within 3 to 5 days post-exposure. Antibodies to varicella-zoster virus are revealed about one week after immunization, but protection often occurs even after an exposure. The development of live attenuated varicella vaccine paved the way for herpesvirus vaccine development. Nevertheless, effective vaccines against herpes simplex virus remain deceptive. Currently, it is possible to distinguish between the Oka strain and the wild type by using polymerase chain reaction (PCR) without having to resort to isolation of the virus. Which exact mutations in vaccine strain are associated with attenuation have not yet been demonstrated. Varicella has an average incubation period of two weeks. Only one serotype of varicella zoster virus (VZV) is identified. VZV is latent in the dorsal root ganglia, cranial nerve ganglia, and enteric ganglia following vaccination and chickenpox. The identifying of latent VZV infection in enteric ganglia is significant because reactivation in enteric ganglia may contribute to several gastrointestinal diseases, such as gastric ulcers, colonic pseudoobstruction (Oglivie’s syndrome), and achalasia.

Reactivation of VZV can contribute to herpes zoster (Shingles). Herpes zoster is usually a painful disease associated with a long-term neuropathic pain syndrome, called “postherpetic neuralgia”. VZV usually does not cause severe illness. Nevertheless, many complications are associated with VZV, such as pneumonia, encephalitis, meningitis, stroke or vasculopathy, and retinal involvement. Annually, estimated 140 million cases of VZV are identified. Severe complications (hospitalization) was found in about 4.2 million people and around 4,200 individuals were annually dead worldwide. Live attenuated varicella vaccine was licensed in the United States in 1995 and now is licensed for routine use in many countries in the world.

In conclusion, varicella vaccines are the first exactly successful preventive measure against varicella virus. The prevention of varicella zoster in persons who already have latent wild-type-virus infection due to past natural VZV infection is a potentially promising goal that is achieved with either inactivated or live vaccine.