Dermatologic Diseases Associated with Human Herpesvirus Type 6 and 7

Attapon Cheepsattayakorn1,2* and Ruangrong Cheepsattayakorn3

110th Zonal Tuberculosis and Chest Disease Center, Chiang Mai, Thailand
25th Office of Disease Prevention and Control, Ratchaburi, Department of Disease Control, Ministry of Public Health, Thailand
3Department of Pathology, Faculty of Medicine, Chiang Mai University, Chiang Mai, Thailand

*Corresponding Author: Attapon Cheepsattayakorn, 10th Zonal Tuberculosis and Chest Disease Center, 143 Sridornchai Road Changklan Muang Chiang Mai 50100 Thailand.

Received: January 01, 2017; Published: March 21, 2017

In 1990, human herpesvirus type 7 (HHV-7) was discovered by June et al whereas human herpesvirus type 6 (HHV-6) or human B-lymphotropic virus (HBLV) was discovered by Gallo, et al in 1986. They identified cytopathic effects in cultures of activated CD4+ T cells isolates from a healthy person and contributed to further ultrastructural and genetic characterization of HHV-7. About 20 - 75% of nucleic acid homology depending on the genes of HHV-7 and both variants of HHV-6 (HHV-6A, HHV-6B) are closely related. CD4+ T cells are the major cell type infected by both HHV-6 and HHV-7. HHV-6 most commonly infects infants of ages 3 to 6 months. Most of the United States population at the age of 3 and 5 years has been infected with HHV-6 and HHV-7, respectively. HHV-7 infection is also identified among middle-age adults. Both HHV-6 and HHV-7 are transmitted via the respiratory route. Primary infection with HHV-6 or HHV-7 is most often asymptomatic, but can cause a roseola-like illness. Usually, there is no obvious signs of upper respiratory tract infection. Coin-sized erythematous macules or slightly elevated papules on the head and neck are characterized in the rashes of the roseola, particularly HHV-6B subtype. HHV-6 can be activated from latency by HHV-7 reactivation with unknown mechanism. HHV-6A has been postulated as a cofactor in the HIV disease progression. HHV-7 has been reported in immunocompromised persons contributing to widespread multi-organ infection, causing encephalitis, pneumonitis, and hepatitis. HHV-7 may also play an important role in persons with a genetic profile of susceptibility to Graves' disease. In 1997, HHV-7 DNA in the tissue of patients with pityriasis rosea (PR), a common papulosquamous disease in healthy young adults was detected by Drago., et al. Increased serum levels of the interferon-gamma is also demonstrated in PR patients. These findings indicate that HHV-7 reactivation is occurred during PR. Some previous studies revealed a possible association between HHV-7 and PR by detection of HHV-7 DNA in 47% of plasma samples and detection of increasing HHV-7 antibody in a few PR patients. Detection of both HHV-6 and HHV-7 DNA by sensitive nested polymerase chain reaction (PCR) in skin, saliva, cell-free serum, and cell-free plasma from most PR patients has been reported.

Presently, the association between HHV-7 and PR is confirmed or not, should not be treated with antiviral therapy. Acyclovir and its derivatives, unfortunately have little antiviral activity against both HHV-6 and HHV-7. In conclusions, further studies are needed to identify the association of herpesviruses and the etiologies of both PR and severe drug-induced hypersensitivity reactions.

Volume 2 Issue 6 March 2017
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