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Editorial

Dermatologic Diseases Associated with Human Herpesvirus Type 6 and 7

Attapon Cheepsattayakorn[1, 2]* and Ruangrong Cheepsattayakorn[3]

[1]10th Zonal Tuberculosis and Chest Disease Center, Chiang Mai, Thailand

[2]5th Office of Disease Prevention and Control, Ratchaburi, Department of Disease Control, Ministry of Public Health, Thailand

[3]Department 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.

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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 erythrematous 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 antiherpesviral therapy. Acyclovir and its derivatives, unfortunately have little antiherpesviral 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.

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