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Recent Developments in the Pathology of Herpes Virus

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Description

The histology of herpes infections is very distinctive. The low power pattern of a typical lesion is of an intraepidermal blister. The key feature is acantholysis with solitary keratinocytes within the blister cavity. Keratinocytes will show nuclear changes in viral infection. These included margination of the nuclear chromatin, multinucleation and nuclear inclusions. Virus inclusions are small pink deposits with a clear halo in the core. If they are present in a herpes virus infection and are present with other major changes in the infection, they are known as cow dry type A inclusions. Cowdry type B inclusion bodies are associated with other infections such as poliovirus and there are no other nuclear changes in herpes infections.

Herpes simplex virus (HSV) infection requires close contact between a person who is susceptible to infection and who is actively shedding the virus or body fluids that contain the virus. Viral shedding occurs during the period of primary infection, subsequent recurrence, and asymptomatic viral shedding. Contact during these periods should be mucous membranes or open skin or frayed skin. After exposure, primary HSV infection is established at the point of contact. The viral envelope fuses with the cell membranes of the skin and mucous membranes, and HSV DNA is integrated into the cell nucleus.

HSV Glycoprotein C protects the viral envelope and aids in viral invasion. Recognition of HSVDNA by Toll-like receptors leads to activation of the innate and adaptive immune system and production of interferon gene products. Viral suppression of the host cell's immune response and subsequent bypass of the immune system is achieved by complex interactions between the HSV virion protein product and the immune system. In particular, the HSV protein Virion Host Shutoff (VHS) is produced in the early stages of infection and suppresses host cell responses. In addition, glycoprotein C binds to complement C3b, inhibits complement-mediated immunity, and inhibits antibody neutralization. The

HSV invades and replicates neurons, epidermal and dermal cells. Villions migrate from the initial site of infection of the skin or mucous membranes to the sensory dorsal root ganglion, where the incubation period is established. Latent states are created by the shutdown of the neuron's HSV genome, and reactivation can occur during periods of stress. Early changes in vacuolar formation in the cytoplasm can be observed along the basal keratinocytes. When cells swell and separate, the cytoplasm becomes eosinophilic, especially in multinucleated cells. Inflammatory infiltrates are mixed, mainly lymphocytes and neutrophils, often interspersed with eosinophils.

Coxsackie virus infection / hand-foot-and-mouth disease: Blisters show intraepidermal vesicle formation and spinal lysis without nuclear inclusions or polynuclearization. Pemphigus vulgaris: Clinically, these conditions are rarely confused. Suprabasal spine melting is observed with vesicle formation in advanced lesions, and a small number of spiny melting cells are found in this space. There are no virus changes. However, it should be noted that disseminated herpes simplex virus infection can complicate pemphigus vulgaris. In difficult cases, immunofluorescence testing is usually the definitive, but additional special testing described above can be used if desired. It is worth noting that many diseases have been reported at sites of herpes zoster infection prior to these include chronic lymphocytic leukemia, granuloma goby, lichen planus, keloid scars, vasculitis, sarcoids, morphia, lymphoma, and skin cancer. The acute changes seen in a viral infection are usually unclear.

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