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Viral infections of the folds (intertriginous areas)

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Abstract Viruses are considered intracellular obligates with a nucleic acid, either RNA or DNA. They have the ability to encode proteins involved in viral replication and production of the protective coat within the host cells but require host cell ribosomes and mitochondria for translation. The members of the families Herpesviridae, Poxviridae, Papovaviridae, and Picornaviridae are the most commonly known agents for the cutaneous viral diseases, but other virus families, such as Adenoviridae, Togaviridae, Parvoviridae, Paramyxoviridae, Flaviviridae, and Hepadnaviridae, can also infect the skin. Though the cutaneous manifestations of viral infections are closely related to the type and the transmission route of the virus, viral skin diseases may occur in almost any part of the body. In addition to friction caused by skin-to-skin touch, skin folds are warm and moist areas of the skin that have limited air circulation. These features provide a fertile breeding ground for many kinds of microorganisms, including bacteria and fungi. In contrast to specific bacterial and fungal agents that have an affinity for the skin folds, except for viral diseases of the anogenital area, which have well-known presentations, viral skin infections that have a special affinity to the skin folds are not known. Many viral exanthems may affect the skin folds during the course of the infection, but here we focus only on the ones that usually affect the fold areas and also on the less well-known conditions or recently described associations.

Introduction

Viruses are considered intracellular obligates with a nucleic acid, either RNA or DNA. They have the ability to encode proteins involved in viral replication and production of the protective coat within the host cells but require host cell ribosomes and mitochondria for translation. The members of the families Herpesviridae, Poxviridae, Papovaviridae, and Picornavirida are the most commonly known agents for the cutaneous viral diseases, but other virus families, such as Adenoviridae, Togaviridae, Parvoviridae, Paramyxoviridae, Flaviviridae, and Hepadnaviridae, can also infect the skin.[1–4]

Viruses may reach the skin by direct inoculation or by spreading from other locations. The cutaneous manifestations of viral infections range from the mild to the severe and from those causing skin infection localized at the site of inoculation to those with associated systemic disease.[1–4]

Though the cutaneous manifestations of viral infections are closely related to the type and the transmission route of the virus, viral skin diseases may occur in almost any part of the body. In addition to friction caused by skin-to-skin touch, skin folds are warm and moist areas of the skin that have limited air circulation. These features provide a fertile breeding ground for many kinds of microorganisms including bacteria and fungi.[5] In contrast to specific bacterial

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and fungal agents that have an affinity for the skin folds, except for viral diseases of the anogenital area, which have well-known presentations, viral skin infections that have a special affinity to the skin folds are not known. Many viral exanthems may affect the skin folds during the course of the infection, but here we will only focus on the ones that usually affect the fold areas and also on the less well-known conditions or recently described associations.

Herpesvirus infections

The herpesvirus group consists of double-stranded DNA viruses. More than 80 herpesviruses have now been identified; only eight of them are known to infect humans. These are herpes simplex virus (HSV) 1, HSV-2, varicella-zoster virus (VZV), Epstein-Barr virus (EBV), cytomegalovirus (CMV), human herpesvirus (HHV) 6, HHV-7, and HHV-8.[3,6,7]

The characteristic feature of herpesvirus infection is the ability to infect epithelial mucosal cells or lymphocytes. Primary infection is the infection of previously seronegative host by HSV types and is often subclinical.[3] The characteristic feature of the herpesviruses infections is the absence of virus elimination after clinical recovery from primary infection; in fact, the virus in the latent form persists during the lifetime of the patient.[3,6] Usually, recurrent HSV is not a real reinfection, but rather it is the result of a viral reactivation.[3,6–8]

Genital herpes

Genital herpes is the most common cause of genital ulceration in the developed countries.[7–10] Genital herpes is transmitted via genital-to-genital and orogenital contact, and a number of risk factors for the acquisition of the virus, including age, sex, ethnic group, socioeconomic status, number of sexual partners, age at first intercourse, and a history of previous sexually transmitted infections, have been reported.[11,12] Classically there are two types of HSV, which are classified by antigenic properties: HSV-1 and HSV-2. In general, HSV-1 is acquired during childhood, transmitted via nongenital person-to-person contact, and is associated with facial infections, whereas HSV-2 is associated with genital infections and indicates the onset of consensual sexual activity.[3,6–13] It has long been recognized that HSV-2 is the major cause of genital herpes, but recent studies have found that the frequency of HSV-1–related herpes genitalis has increased considerably.[3,11]

In patients with symptomatic primary HSV infection, the clinical signs and symptoms may range from very mild to very severe.[6,7] The incubation period is approximately 2 to 14 days for the primary genital infection.[7,10] Classically the infection is preceded by genital pain, itching, or burning sensation lasting up to 24 hours.[6] Systemic manifestations, such as headache, fever, and swollen regional lymph nodes; dysuria; urinary retention; vaginal or penile discharge; and groin sensitivity, can accompany this. The lesions may begin as small, clear, short-lived painful vesicles with an erythematous base, immediately followed by shallow yellow-gray ulcers.[3,6,7,10] These superficial ulcers erode quickly, especially on mucosal surfaces, and heal without scarring. New crops of vesicles continue to develop for the first 1 to 2 weeks of the disease.[3,6,7,10] The lesions of the primary genital infection, but not the viral shedding, last for 2 to 6 weeks.[3] In men, lesions are located on the shaft and glans of the penis, whereas in women they are usually located on the labia minora and urethra meatus.[6]

Recurrent genital HSV infections are generally less severe than the primary infection, with fewer lesions and a slightly shorter duration of symptoms.[3,7] The number and the frequency of recurrent infections are closely related to sex, virus type, and severity of the primary infection.[7] Recurrent genital herpes is most common in the first year after the primary infection, but the rate of symptomatic recurrence tends to decrease over time.[6] Recurrences may also be triggered by overexposure to the ultraviolet radiation, physical and psychological stress, skin irritation in the genital area, menstruation, and pregnancy.[3]

Though humoral and cell-mediated immunities do not fully protect against reinfection or recurrent disease,[3] a deficit in HSV-specific cell-mediated immunity results in delay in the normal rapid clearing of HSV from mucocutaneous lesions.[13] In immunocompromised patients, HSV infection may be more extensive and chronic and may become necrotic, and there is a possible risk for dissemination of the virus.[3,6,13–16] Central nervous system involvement may occur in HIV-positive patients.[13]

Recent literature describes two uncommon presentations of cutaneous herpes infections.[14–16]

Verrucous genital herpes

Although atypical presentations of genital herpes are more commonly described among immunocompromised patients, verrucous genital herpes infection has also been reported in an immunocompetent patient.[14] The patient was a 60-year-old man who presented with multiple rapidly developing, well-demarcated, round, pink exophytic papules and nodules coalescing into larger plaques exhibiting superficial erosions and ulcerations. Lesions were located on the scrotum, inguinal folds, and inner thighs. This case report indicates that HSV infection should be considered in the differential diagnosis of persistent genital verrucous lesions in both immunocompetent and immunocompromised patients.

Knife sign of the herpetic infections

Painful deep linear intertriginous ulcerations and fissures have recently been described as a manifestation of cutaneous