HSV-2, HPV, CMV,

and Molluscum contagiosum virus

UG module

Microbiology lecture 8

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Genital herpes

Etiology: most commonly HSV-2

Transmission:

Direct contact with mucosal tissue or secretions of another infected person

HSV-2 is mostly spread through genital contact and should, therefore, raise suspicion for sexual abuse if found in children.

Pathophysiology

- Inoculation: The virus enters the body through mucosal surfaces or small dermal lesions.
- Neurovirulence: The virus invades, spreads, and replicates in nerve cells.
- Latency: After primary infection, the virus remains dormant in the ganglion neurons (Sacral ganglion).
- Reactivation: triggered by various factors (e.g., immunodeficiency, stress, trauma) → clinical manifestations.

Type of infection:

- Primary infection: Mostly asymptomatic (up to 80% of cases, but virus is still shed).
- Reactivation of infection: Frequency and severity vary individually; symptoms are usually less severe than in primary infection. Often at the same site as primary infection

Clinical features

Affected individuals are often asymptomatic or have mild symptoms but may still be at risk of transmission.

Skin lesions: may be present in both initial and recurrent infection.

The patient might be presented with redness, swelling, tingling, pain, and pruritus, then grouped erythematous vesicles that progress to painful ulcers in the anogenital area appear.

Primary infection:

- Genital tract: skin lesions in the anogenital area, cervicitis, white, thick, and/or foul-smelling vaginal discharge
- Urinary tract: dysuria, urethritis

- Associated symptoms: fever, headaches, myalgias, malaise, tender bilateral inguinal lymphadenopathy

Recurrent infection

- pain or tingling in the genitals, legs, buttocks, and/or hips
- Skin lesions are usually unilateral, less painful, and of shorter duration than in the initial infection.

Diagnostics: Make a clinical diagnosis of HSV infection or reactivation.

Confirm diagnosis with PCR and/or viral culture in patients with suspected infection or reactivation regardless of symptoms.

Treatment: Acyclovir

Antiviral treatment effect: Decrease in duration and severity of infection, Reduction of viral shedding, However, recurrence cannot be prevented.

Human papillomavirus infection (HPV)

Human papillomavirus

Double-stranded, circular, nonenveloped DNA virus with an icosahedral capsid

Low-risk HPV types 6 and 11:

- Anogenital warts (condylomata acuminata)
- Mild cervical cell abnormalities
- Tumors of nongenital mucosal membranes (e.g., respiratory tract, oral cavity)

High-risk HPV types 16, 18, 31, and 33

- Cervical cancer (responsible for 70% of cases)
- High risk of anogenital, oral, and oropharyngeal squamous cell carcinoma

HPV types 1, 2, and 4: cause skin warts, such as common warts (verruca vulgaris) and plantar warts

Route of transmission

Transmission occurs between two epithelial surfaces.

Close personal contact: cutaneous warts

Sexual contact: anogenital lesions

Pathogenesis

HPV expresses the following oncoproteins E6 and E7 that facilitate cellular transformation by inactivating tumor suppressor proteins, such as p53 and retinoblastoma (Rb), leading to uncontrolled cell proliferation and the development of HPV-related cancers.

Genital intraepithelial neoplasms

Pathogen: HPV types 16 and 18

Classification:

- Squamous intraepithelial lesion: low-grade or high-grade, such as Cervical intraepithelial neoplasia, Penile intraepithelial neoplasia, and Anal intraepithelial neoplasia.
- Squamous cell carcinoma such as Cervical cancer, Carcinoma of the penis, and Anal cancer.

Condylomata acuminata (anogenital warts)

Pathogen: HPV types 6 and 11 (responsible for ~ 90% of genital warts)

Location: ♀: vulvar, cervix, anal region,

♂: glans penis, foreskin, urethra, anal region

Clinical features: Exophytic, cauliflower-like lesions. Often asymptomatic; may cause pruritus, tenderness.

Diagnostics: Visual inspection, Application of 5% acetic acid turns lesions white (not a specific finding).

Treatment of anogenital warts:

- Pharmacotherapy: local cytostatic treatment
- Cryotherapy: freezing external warts with CO2, or N2

Flat condylomata

Pathogen: particularly HPV types 3 and 10

Clinical features: flat, white-brown, slightly elevated, scattered plaques in the anogenital region

Diagnostics: visual inspection

Treatment: Curettage or laser surgery

Regular checks: necessary because of the high risk of malignancy

Non-anogenital manifestations:

Common warts → Lesions are plaques or papules, Skin-coloured or whitish usually firm, often with a rough and scaly surface, located on the elbows, knees, fingers, and/or palms.

Plantar warts → Rough, hyperkeratotic lesions on the sole of the foot often grow inwardly and cause pain while walking.

Flat warts → Multiple small, flat patches or plaques localized on the face, hands, and shins.

Treatment

There is no treatment for the infection itself. In most cases the infection clears up without any treatment.

For the treatment options of HPV–related anogenital warts, routine clinical monitoring is important.

Molluscum contagiosum

Pathogen: a DNA poxvirus (molluscum contagiosum virus)

Transmission:

- Direct skin contact (contact sports, sexually transmitted)
- Autoinoculation (scratching or touching lesion, e.g., while shaving)
- Fomites (e.g., on bath sponges/towels)

Physical examination: single or multiple lesions in healthy patients; especially widespread in immunocompromised patients.

Nontender, skin-coloured, pearly, dome-shaped papules with central umbilication (individual lesions may also be painful or pruritic)

Predilection sites:

In children: face, trunk, and extremities (e.g., axilla, antecubital and popliteal fossa)

In adults: lower abdomen, groin, genitalia, and proximal thighs.

Treatment: Spontaneous remission of the lesions usually happens within a few months; thus, treatment is often unnecessary.

If treatment is indicated (e.g., for sexually transmitted molluscum contagiosum), cryotherapy with liquid nitrogen is usually the first treatment option.

Cytomegalovirus infection

Pathogen: cytomegalovirus (CMV, human herpes virus 5, HHV-5)

Transmission:

- Blood transfusions
- Sexual transmission
- Transplacentally (highest risk during the third trimester of pregnancy)
- Perinatal transmission (e.g., contact with contaminated blood/vaginal secretions during delivery or breastfeeding)
- Body fluids (e.g., respiratory droplets, saliva, urine, genital secretions)

Clinical features:

CMV infection is usually asymptomatic. Severe manifestations occur in patients with immunocompromise (e.g., following organ transplantation, AIDS).

Immunocompetent patients: > 90%: asymptomatic course

< 10%: CMV mononucleosis

Fever, malaise, myalgia/arthralgia, fatigue, headache

Less common: sore throat, cervical lymphadenopathy, hepatomegaly, splenomegaly

Immunocompromised patients

- Asymptomatic CMV infection: evidence of viral replication (e.g., viral DNA or antigen in serum) but no symptoms
- Viral syndrome: malaise and fever with leukopenia and/or thrombocytopenia in individuals with a positive serum CMV antigen
- CMV pneumonia: interstitial pneumonitis
- CMV retinitis
- CMV esophagitis and/or CMV colitis
- CMV hepatitis: prolonged malaise and fever with mild transaminitis
- CMV encephalitis: impaired cognitive function, neurological deficits
- Adrenal insufficiency