Common Cutaneous Infectious And Infestations PART 2

Objectives :

- To be familiar with common bacterial, viral, mycobacterial, fungal,and parasitic infections of the skin.
- To be familiar with presentation and management of common skin infections.
- To be familiar with skin infestation, their clinical features, prevention and treatment.
- The main reference is the TEXTBOOK..

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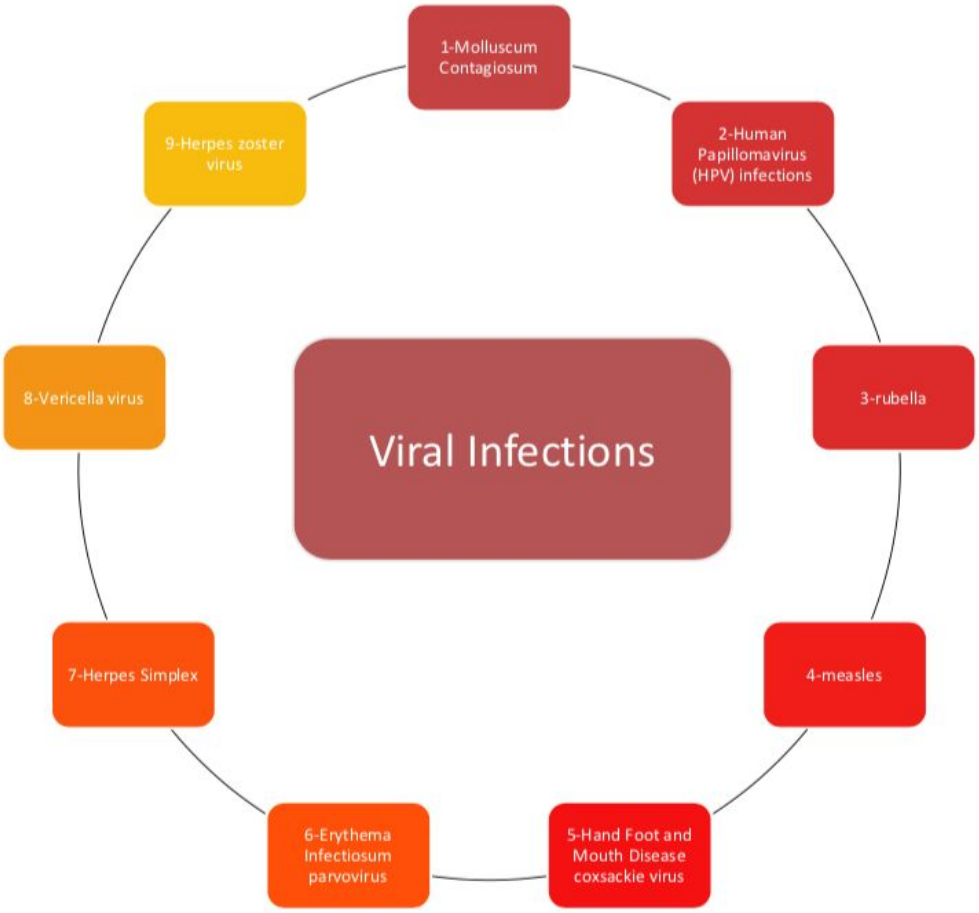
Revised by:

***Sources**: FITZPATRICK color atlas +433 male + 434 + doctors slides and notes*

[Color index : Important | Notes | Extra]

Part 3: Viral Infections



•Primary infections with many viruses cause acute systemic febrile illnesses and exanthems, are usually **self-limited**, and convey lifetime immunity.
•Herpes virus infections often have asymptomatic primary infection but lifelong latent infection. With immunosuppression can become active and cause disease.

1-Molluscum Contagiosum

ORGANISM	•**Molluscum Contagiosum virus.** •**Self limited** viral infection
TRANSMISSION	**skin to skin contact.**
EPIDEMIOLOGY	•More **common in children**, in adults mostly sexually transmitted.
CLINICAL MANIFESTATIONS	•Multiple scattered skin colored umbilicated papules and nodules. •Autoinoculation can occur by scratching or touching the lesions. •Can be widespread in patients with atopic dermatitis.
INVESTIGATIONS	•Histopathology: infected cells contain large **intracytoplasmic inclusions called Henderson-Pattterson bodies.**
COURSE	•Often persists for 6 months then undergo spontaneous regression without scarring.
TREATMENT	**curettage**, cryotherapy, electrodessication, and cantharidin. **Explained next slide**

Curettage: is the use of a curette to remove tissue by scraping or scooping.
Cryotherapy: any treatment that involves the use of freezing or near-freezing temperatures
electrodessication: simple office-based technique where an electrical current is used to remove specific skin lesions such as sebaceous hyperplasia
Cantharidin an extremely hazardous substance

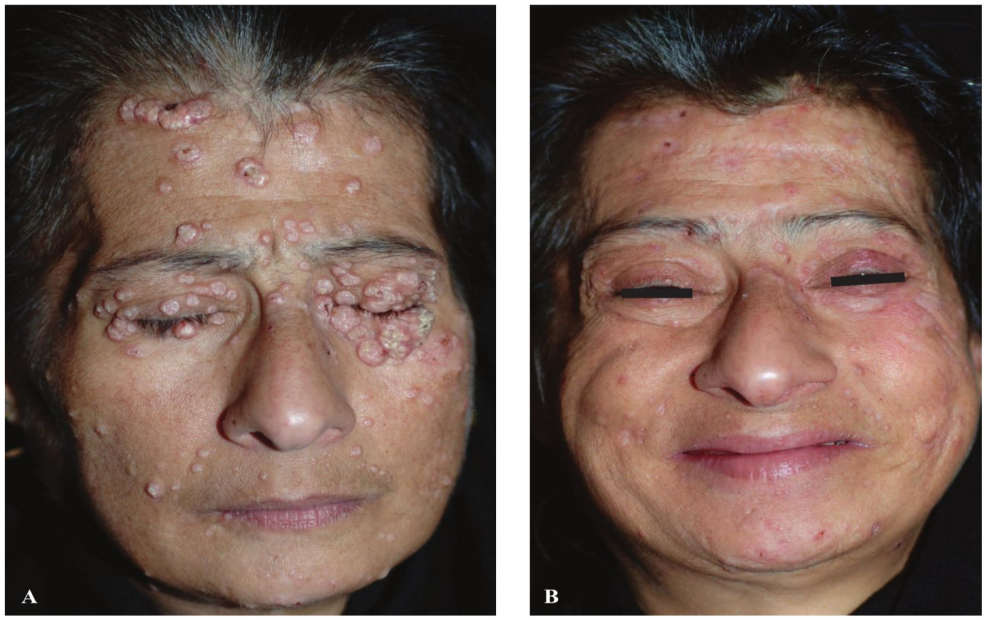


FIGURE 27-2 **Molluscum contagiosum: face** **(A),** large lesions on the face of a HIV+ woman.

2-Human Papillomavirus (HPV) infections

ORGANISM	•**Double stranded DNA** viruses that infect squamous epithelia of skin and mucus membrane.
TRANSMISSION	•Transmitted by **skin to skin contact.**
CLINICAL MANIFESTATIONS	•depends on HPV type. •Genital warts in mothers during delivery can be transmitted to neonates resulting in genital warts and respiratory papillomatosis. •**HPV 16,18,31,33 has high malignant potential** and could lead to cervical carcinoma in women.
RISK FACTORS	•Immunosuppression, organ transplantation and HIV, **increase the incidence and severity of warts.**

TYPES:

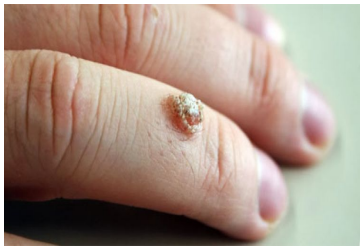
A-Verruca Vulgaris
B-Plantar Warts
C-Flat Warts
D-Epidermodysplasia Verruciformis
E-Common Warts
F-Genital Wart

A-Verruca Vulgaris

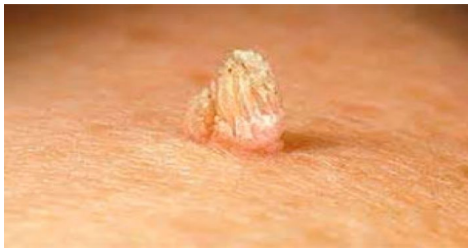
ORGANISM	•Mostly caused by HPV 1, 2.
CLINICAL MANIFESTATIONS	•Firm papules from 1-10 mm hyperkeratotic, clefted surface, wit vegetations (warty), **with characteristic red or brown-black dots.** •Inoculation by scratching lead to linear arrangement.
DDx	Molluscum Contagiosum, seborrheric keratosis, actinic keratosis, SCC.

1-Annular warts at sites of previous therapy.
2-Filiform warts with small bases.
3-Butcher warts, large cauliflower-like lesions on hands of meat handlers.

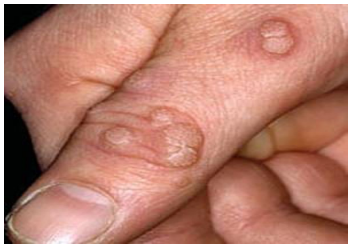
1-ANNULAR



2- FILIFORM

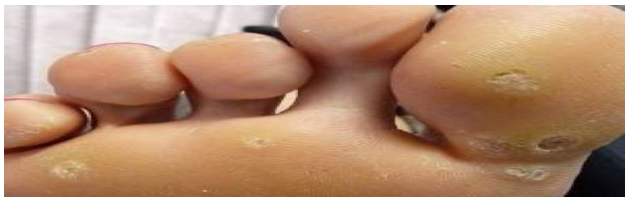
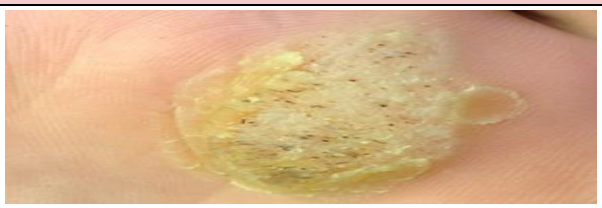


3-BUTCHER WART



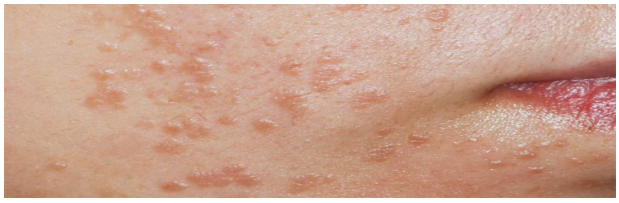
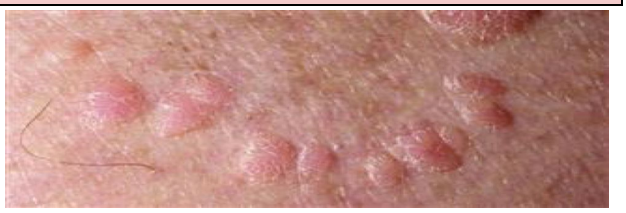
B-Plantar Warts

ORGANISM	•HPV 1, 2
CLINICAL MANIFESTATIONS	•**Hyperkeratotic plaques** with **brown dots** on plantar surface,**might be tender when walking.** •Mosaic warts if multiple papules coalesce to form large plaque, difficult to treat.
DDx	•callus an area of thickened skin •corn is a specially shaped callus of dead skin

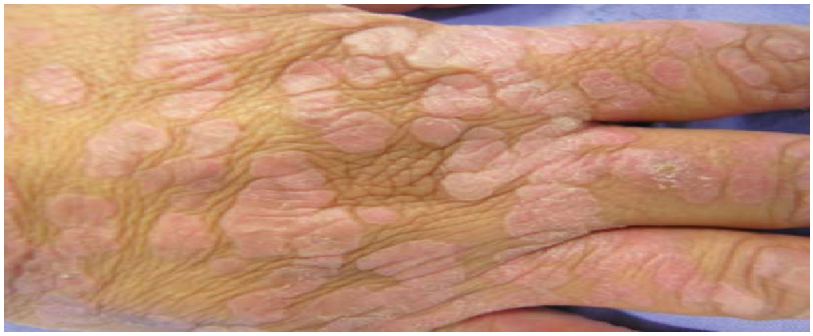
B-Flat Warts

ORGANISM	•HPV 3, 10
CLINICAL MANIFESTATIONS	•Small skin colored or light brown flat papules 1-5 mm in size mostly on face, beard area, dorsum of hands.
DDx	syringoma, Molluscum Contagiosum

D-Epidermodysplasia Verruciformis

ORGANISM	•HPV 5,8, **Autosomal recessive.**
CLINICAL MANIFESTATIONS	•first occur at 5-7 years. •Flat topped papules, skin colored to light brown, could be numerous on face, dorsa of hands, arms, legs, anterior trunk. •Premalignant and malignant lesions mostly SCC on the face in 30-50% of patients.
DDx	Pityriasis Versicolor, actinic keratosis, seborrheic keratosis, BCC,SCC.



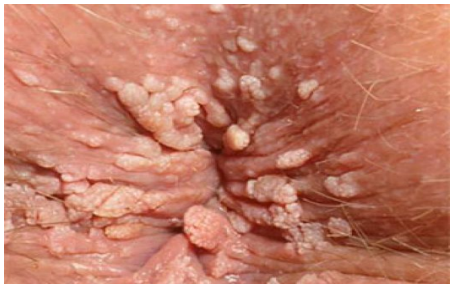
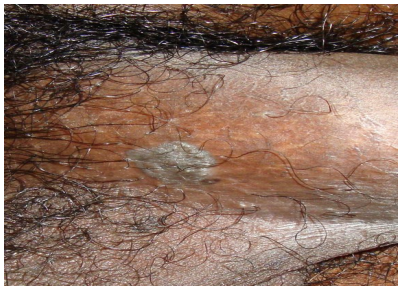
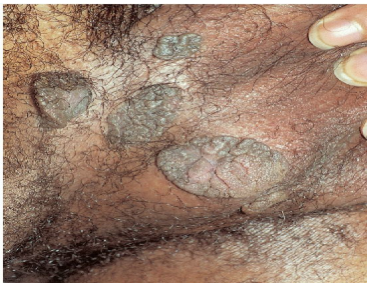
E.Warts

INVESTIGATIONS	•Histopathology: acanthosis, papillomatosis, hyperkeratosis. Vacuolated cells, vertical tiers of parakeratotic cells, and foci of clumped keratohyaline granules.
COURSE	•Warts in immunocompetent individuals usually resolve spontaneously. In immunocompromised patients they persists and tend to resist therapy.
TREATMENT	•salicylic acid 27-40%, cryotherapy, Electrocautery, laser, surgery. •DPCP, candida antigens, bleomycin injection.

E.Genital Warts

ORGANISM	•HPV 6, 11 are the most common, 16,18,31,33 has high malignancy potential. •Laryngeal papillomas associated with HPV 6,11.
TRANSMISSION	•sexual contact, mother to neonates. •Incubation period 3-30 weeks. Mostly 10 weeks.
CLINICAL MANIFESTATIONS	•Usually asymptomatic, four clinical types: 1-small papular,2-cauliflower-floret, 3-keratotic, 4-flat topped papules most common on cervix.
RISK FACTORS	number of sexual partners, infection with other STDs.
DDx	Pityriasis Versicolor, actinic keratosis, seborrheic keratosis, BCC,SCC.

INVESTIGATIONS	**Pap smear** for women with warts. Screening for STDs for all patients.
COURSE	**•Highly infectious,** if left untreated my resolve on their own, remain unchanged, or grow. (infection may persist for life). •After regression subclinical•In pregnancy warts may increase in size and number.
TREATMENT	•imiquimod 5% cream, podophylox 0.5% solution, podophyllin10-20% (applied at the clinic), cryotherapy, Electrocautery, surgery. •Prevention: HPV vaccine. •No therapy will eradicate HPV.

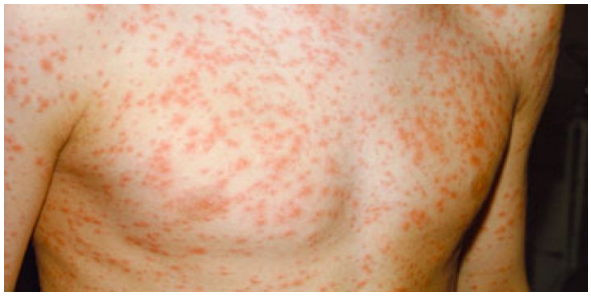
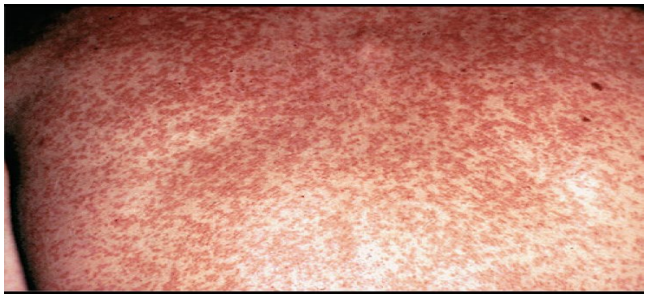




Systemic viral infections with exanthems

1. **Rubella**
2. **Measles**
3. **Hand and foot**
4. **Erythema infectiosum**

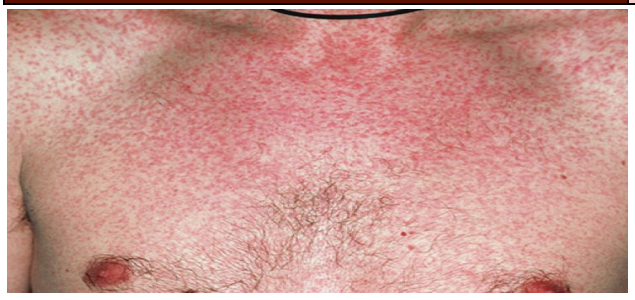
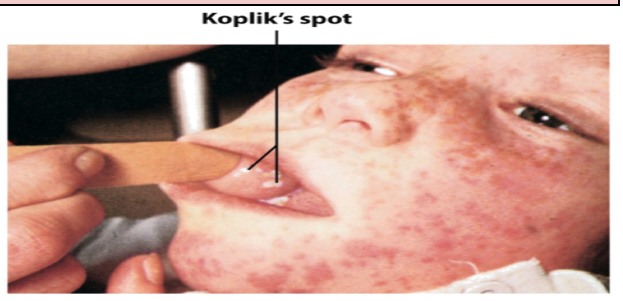
3-Rubella

ORGANISM	•Rubella virus an RNA togavirus.
AGE	•Children <15 years old.
TRANSMISSION	•through inhalation of aerosolized droplets.
CLINICAL MANIFESTATIONS	•Pink macules, papules initially on forehead spreading to face and trunk that fades after 3 days. Petechiae on soft palate and lymphadenopathy. **•Prodrome usually absent.** •Low grade fever, malaise, headache, mild URT symptoms. •Arthritis in adults, splenomegaly.
RISK FACTORS	•Congenital rubella syndrome: congenital heart defects, cataracts, microphthalmia, microcephaly, hydrocephaly, and deafness.

4-Measles

ORGANISM	•RNA morbillvirus.
TRANSMISSION	•respiratory droplets. •Incubation period 10-15 days.
CLINICAL MANIFESTATIONS	•Fever, malaise, coryza, vomiting, diarrhea, photophobia, conjunctivitis and periorbital edema. These symptoms subside with appearance of rash. •On the 4thday, erythematous macules and papules appear on the forehead, behind the ears then face, trunk, extremities, reaching feet by the 3rdday and resolve in 6 days. •Tiny bluish-white spots on red background on the 2ndday of fever on the buccal mucosa **(Koplik spots).** •Generalized lymphadenopathy, splenomegaly.
COMPLICATIONS	•Acute complications in 10%: otitis media, pneumonia, diarrhea, encephalitis, and thrombocytopenia. •Chronic complication: subacute sclerosing panencephalitis.

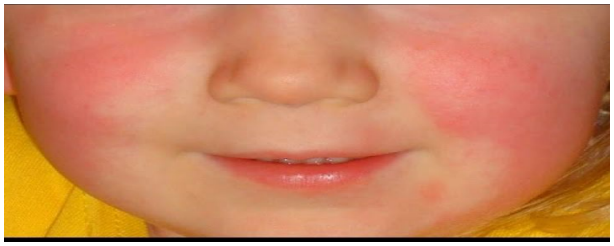
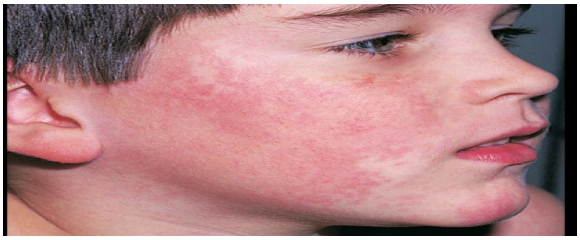



5-Hand Foot and Mouth Disease

ORGANISM	•Coxsackievirus A16 and •Enterovirus 71.
AGE	•First decade of age, late summer or early fall.
CLINICAL MANIFESTATIONS	•Multiple painful mouth ulcers, macules and papules that evolve to vesicles on palms ,soles, sides of fingers, toes and buttocks. •Vesicles can rupture with formation of erosions and crusts. •May be associated with fever, malaise, diarrhea, and arthralgia. •Enterovirus 71. infections have higher morbidity from CNS and pulmonary edema.
TREATMENT	•Self limited, symptomatic treatment.

6-Erythema Infectiosum

ORGANISM	•Human parvovirus B19.
TRANSMISSION	•Transmission by respiratory droplets. •Viremia develops 6 days after intranasal inoculation of the virus, significant bone marrow suppression (aplastic crisis) can occur at this time.
CLINICAL MANIFESTATIONS	•Exanthem begins 18 days with arthralgia. •Fever and adenopathy are more severe in adults. •Numbness and tingling of fingers. •Diffuse erythematous patches on cheeks (slapped cheek) fades over 1-4 days, this is absent in adults. •Erythematous macules, papules that become confluent with reticulated appearance on extensor arms, trunk and neck fade in 5-9 days.

7-Herpes Simplex Virus

ORGANISM	•HSV 1 mostly herpes labialis & •HSV 2 mostly genital herpes.
TRANSMISSION	•skin-skin or skin-mucosa contact. •After primary infection, HSV persists in sensory ganglia for life.
CLINICAL MANIFESTATIONS	•After exposure, the virus replicate in epithelial cells, causing lysis of infected cells, **vesicle formation and local inflammation.**
RECURRENCY ?	•Recurrence with skin or mucosa irritation, UV radiation, menstruation, fever, common cold, immunosuppression. •Recurrent viral shedding can be with or without lesions, and transmission usually occur while shedding.
INVESTIGATIONS	•**Tzanck smear:** fluid from vesicles stained with Giemsa stain will show or multinucleated giant acantholytic keratinocytes. •**Direct flourescent anibody will differentiate between type 1&2.** •**Viral culture,** serum antibodies for previous infections, seroconversion will take 2-6 weeks.

A-Non-Genital Herpes Simplex

CLINICAL MANIFESTATIONS	•Primary infection is often asymptomatic. •Primary herpetic gingivostomatitis is the most common in children. •Grouped vesicles on erythematous base that rupture easily forming erosions and crusts with regional adenopathy.

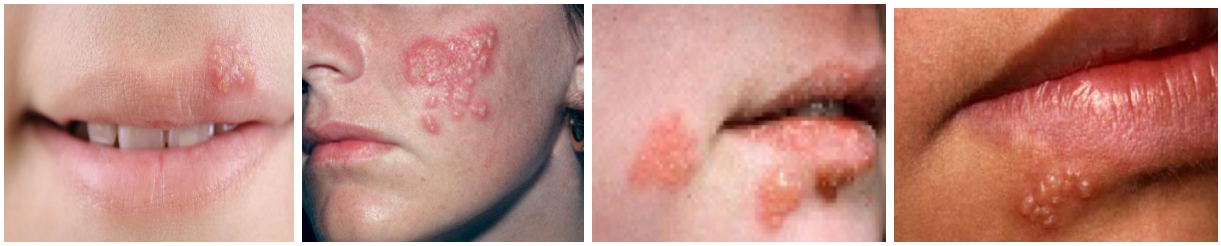
CONT..

RISK FACTORS	other dermatoses with risk of HSV infection are Darier disease, burns, Hailey-Hailey, bullous disorders, ichthyosis.
RECURRENCE	•Recurrent herpetic infection has prodrome of tingling, itching, or burning sensation 24 hours prior to skin lesions.
TREATMENT	•For primary herpes: Acyclovir 400 mg tid for 7-10 days. •Valacyclovir, prodrug with better bioavailability 1 g bid for 7 days. •Famciclovir 250 mg tid for 7 days. •Maintenance therapy in patients with > 6 attacks/year: valaciclovir 500mg daily, acyclovir 400 mg bid, famciclovir 250mg daily. •For recurrence: same doses for acyclovir and valacyclovir for 5 days. , 1500 mg one dose for recurrent herpes labialis.

VARIANTS:

1-Herpetic whitlow: infection of the tip of finger or thumb with painful neuritis in affected finger.

2-Eczema herpeticum: HSV infection in atopic patients with widespread vesicles not grouped,



A-Genital Herpes Simplex

ORGANISM	•HSV 2 mainly,
TRANSMISSION	•skin-skin contact With 70% of transmission during times of asymptomatic HSV shedding.
CLINICAL MANIFESTATIONS	•Most clinical lesions are minor breaks or erosions, abrasions. •Primary infections mostly asymptomatic, erythematous papules evolving to vesicles and erosions that heal in 2-3 weeks. •Inguinal lymph nodes may enlarge.

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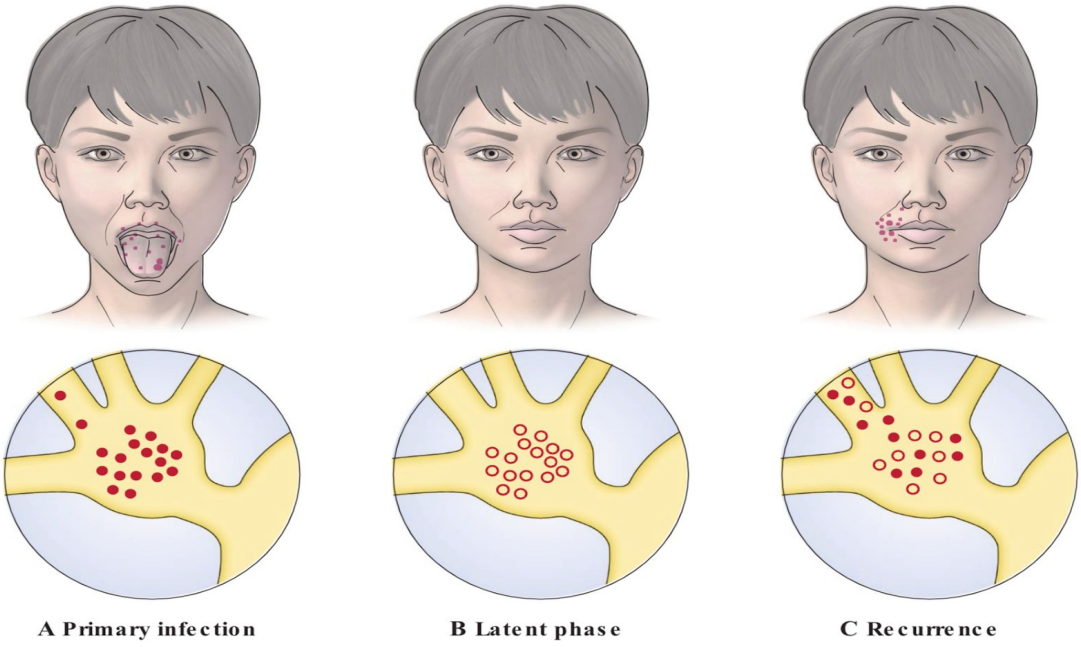
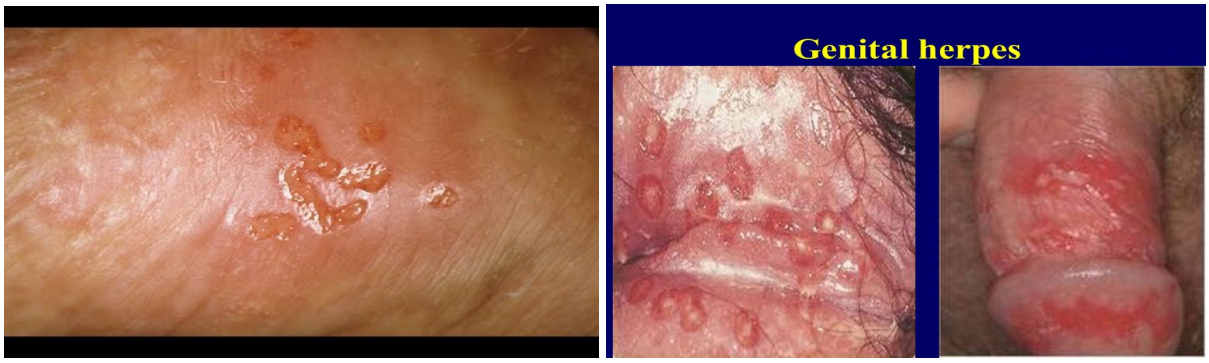
RECURRENCE	•Recurrent genital herpes my present with itching, burning, redness prior to eruption of vesicles.
TREATMENT	•Famciclovir 1g bid for one day for recurrent genital HSV



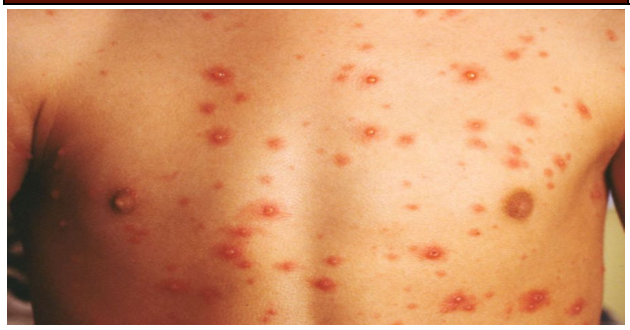


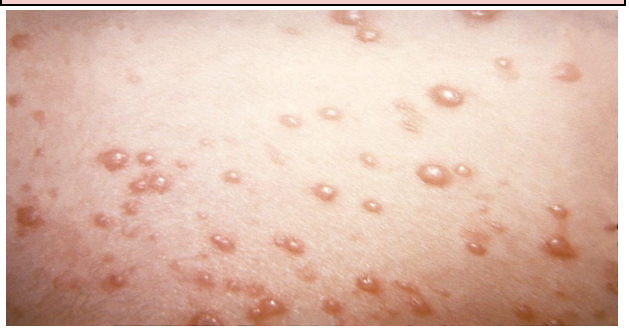
8-Varicella Virus

AGE	•Without immunization 90% of cases before age 10.
TRANSMISSION	•airborne droplets and direct contact. •Patients are contagious few days before the rash and until the last crop of vesicles dry out. •Vaccination is 80% effective.

CONT..

CLINICAL MANIFESTATIONS	•Vesicular lesions occur in successive crops. Few vesicles in children but more numerous in adults. •First lesions are papules that quickly evolve to vesicles with surrounding erythema. Vesicles evolve to pustules and crusted erosions after few hours that heal in 3 weeks leaving punched out scars. •Lesions start on face then trunk and extremities, highest densities on back.
COMPLICATIONS	•In adults pneumonitis, cerebellar ataxia and encephalitis may occur.
TREATMENT	•usually symptomatic treatment. •If oral antiviral therapy is given in the first 24 hours the severity will be decreased. •Neonates: **acyclovir** 10mg/kg/q8h for 10 days. •Children: **acyclovir** 20mg/kg/q6h or **valacyclovir** 20mg/kg/q8h for 5 days. •Immunocompromised adults: **acyclovir** 800mg 5times/day, or **valacyclovir** 1g tid, or **famciclovir** 500 mg tid for 7-10 days.





9-Herpes Zoster Virus

TRIGGERING FACTORS	•Reactivation of varicella virus in those ganglia in which varicella virus has achieved the highest density. • immunosuppression, trauma, tumor, or irradiation.

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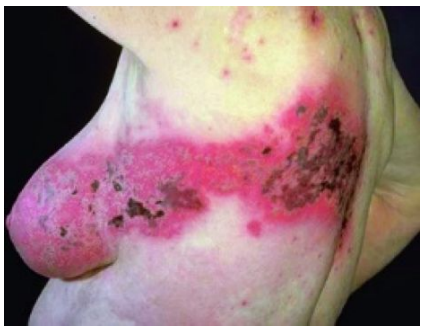
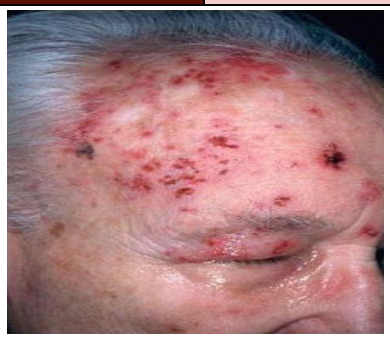
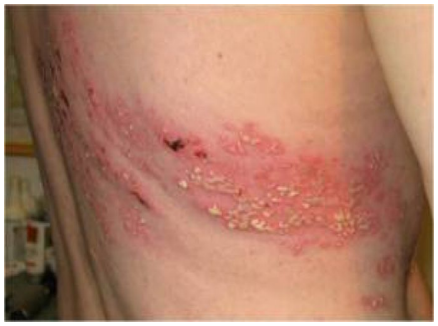
CLINICAL MANIFESTATIONS	•Prodrome with pain, tenderness and paresthesia in the involved dermatome. Flu like symptoms can occur. •Papules evolve to vesicles and pustules in 3-4 days then crusts after 10 days with new vesicles appearing over one week. All on erythematous base with regional adenopathy. Crusts resolve totally after 3-4 weeks. •**Sometimes scars and post-inflammatory Hyperpigmentation persists for years.** •**Thoracic area 50%**, trigeminal 20%, lumbosacral 10%. •**Ophthalmic zoster**: nasociliary involvement **of ophthalmic branch of trigeminal nerve**, early lesions vesicles on tip of nose, may lead to keratitis, conjunctivitis, retinitis, optic neuritis. Has the highest incidence of post herpetic neuralgia. •**Post herpetic neuralgia**: constant severe stabbing or burning pain in 40% of patients above 60 often resolves in 6 months but may persist for years.
TREATMENT	•Vaccine against VZV reduces the incidence of zoster by 50%. •Famciclovir 500mg/8h/7days. •Valacyclovir 1g/8h/7days. •Acyclovir 800mg/5times daily/7-10 days. •In immunocompromised: acyclovir 10mg/kg IV Q8h for 10 days. •In acyclovir resistant: IV foscarnet 40mg/kg IV Q8h till resolution. •For post herpetic neuralgia: gabapentin, pregabalin, tricyclic anti depressant, analgesics, nerve block.

PART 4 PARASITIC INFECTIONS

Parasitic Infections		
1-Pediculis capitis	2-leishmaniasis	3-scabies

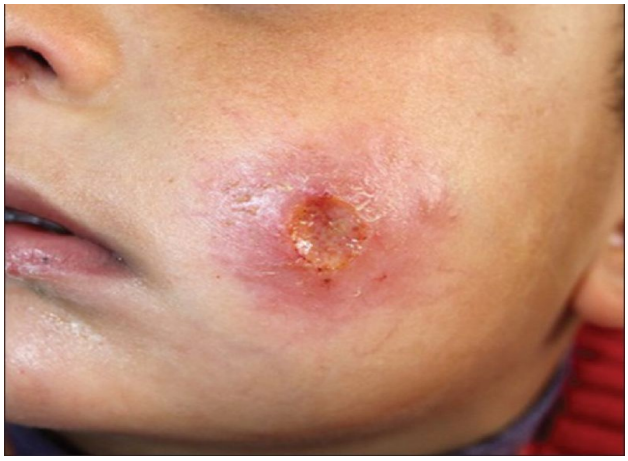
1-Pediculosis Capitis قمل الرآس

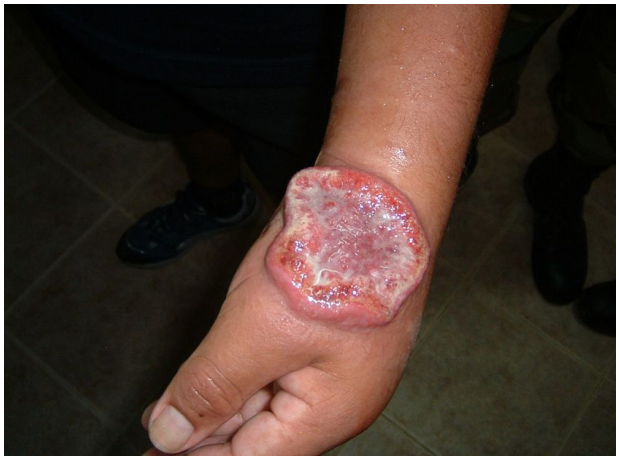
ORGANISM	•Infestation by Pediculus Humanus Capitis 1-2 mm in size.
TRANSMISSION	•**head to head contact**, shared hats, comb, brushes, **pillows**. •Can crawl 23cm/day. lay nits 1-2 mm from scalp. •Lice hatch within 1 week, mature into adult within 1 week. •One female can lay 100 ova in 2 weeks lifetime. Can survive only for few hours off scalp. •New viable eggs are creamy-yellow, empty eggshells are white.
CLINICAL MANIFESTATIONS	•Present as **pruritus** of the back and side of scalp with secondary bacterial infection and regional adenopathy.
TREATMENT	•topical **Permethrin**, malathion, pyrethrin, dimethicone, and Ivermectin(topical or systemic in severe cases)



2-Leishmaniasis

ORGANISM	•Leishmania tropic major Incubation period 1-4 weeks .
EPIDEMIOLOGY	•Common in Alkharj, Alahsa, Alqaseem, Hail, Gizan.
CLINICAL MANIFESTATIONS	•Single or multiple asymptomatic cutaneous papules and nodules at the site of a sand-fly bite that may evolve over weeks to ulcer which heal spontaneously with a depressed scar.
INVESTIGATIONS	•Histopathology: Dermal granulomatous inflammation with prominent lymphocytes, histiocytes with intracellular non-flagellated amastigote.
TREATMENT	•sodium stibogluconate intralesional if single lesion every week till resolution. Or systemically IM 15mg/kg/d for 21 days. •Itraconazole, amphotericin B, paromomycin, miltefosine.





3-Scabies الجرب

ORGANISM	•Epidermal infestation by **mite Sarcoptes scabiei var. Hominis.** •Female life span 4-6 weeks, lays 3 eggs daily, eggs hatch in 4 days.
TRANSMISSION	•skin-skin contact and fomites. •Leave burrow at night and lay eggs during the day. can be alive for 2 days on clothing or bedding. •Mites burrow (DIG) into epidermis shortly after contact no deeper than stratum granulosum (2nd outermost layer)
CLINICAL MANIFESTATIONS	•**Intense pruritus** is the main symptom, hypersensitivity of immediate and delayed type are responsible for other lesions other than the primary burrows. •Secondary changes: excoriations, lichen simplex chronicus, prurigo nodules, PIH, secondary infections, id reaction.
INVESTIGATIONS	•**microscopy** by putting a drop of mineral oil on the burrow, then the burrow is scraped off then placed on microscopic slide to look for mites, eggs and fecal pellets(scybala). •**Histopathology**: female mite with eggs, Spongiosis and vesicle formation. Eosinophilic infiltrate in dermis.
TREATMENT	•bed cleaning. •**Permethrin** 5% cream to whole body for 8 hours or overnight. •**Crotamiton**, sulfur, benzyl benzoate, malathion, Lindane, Ivermectin. •Oral **Ivermectin** 0.2mg/kg single dose. •**Post scabietic itching** may persist after successful treatment represent **hypersensitivity to remaining dead mites and its products.**

•Burrows are skin colored ridges 1cm long with minute vesicle or papule at the end of the tunnel. Mainly on interdigital webs, wrists, penis, buttocks, scrotum. And in infants might involve head and neck. Sometimes there are nodules on scrotum, penis, axillae, and buttocks.