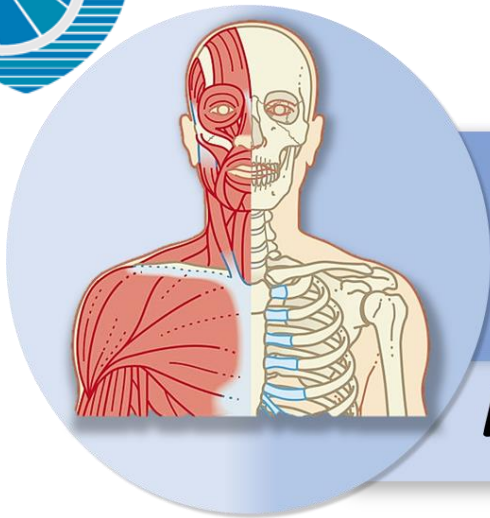




mss system

Microbiology

☒ **Sheet**

☐ **Slide**

Number:

1

Done by:

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Outline

- A review of the previous e-lecture.
 - Viral Skin Infections.
 - 1- HSV → Serious conditions: Eczema Herpeticum, Erythema Multiforme, SJS/TEN.
Not serious conditions: Herpes labialis and genital Herpes.
 - 2- Herpes-caused infections: Herpetic whitlow and Herpes gladiatorum.
 - 3- VZV: Chickenpox and Shingles.
-

E-lecture Review

“The sheet was written based on our first lecture with the Doctor. The e-lecture is not included in this sheet, these are just some points the doctor mentioned as a review”.

- **Macule**: Discoloration of the skin → **Papule**: Raised macule → **Vesicle**: Filled papule.
Bulla: Larger papule → If the bulla breaks, it becomes a **crust**.
- **Ulcer**: Losing part of the epidermis.
- **Cellulitis**: Infection spreading into the subcutaneous tissue.
- **Necrotizing fasciitis**: Infection involving superficial and deep fascia.
- **Myositis**: Infection of the muscles.
- **Osteomyelitis**: Inflammation of the bone.
- Knowing the **type of rash** aids in determining the causative pathogen, whether it is **bacterial, viral** or **fungal**.
- Almost **all** vesicular rashes are **viral** except a few rare ones.

LESION, CLINICAL SYNDROME	INFECTIOUS AGENT
Vesicles	
Smallpox	Variola virus
Chickenpox	Varicella-zoster virus
Shingles (herpes zoster)	Varicella-zoster virus
Cold sores, herpetic whitlow, herpes gladiatorum	Herpes simplex virus
Hand-foot-and-mouth disease	Coxsackievirus A16
Orf	Parapoxvirus
Molluscum contagiosum	Pox-like virus
Rickettsialpox	<i>Rickettsia akari</i>
Blistering distal dactylitis	<i>Staphylococcus aureus</i> or <i>Streptococcus pyogenes</i>

The doctor asked us to memorize it.

- Rashes may be of a **local** problem in the skin, or as a **systematic** manifestation of a disease such as in salmonella.

Viral Skin Infections

A- Herpes Simplex Virus

- Herpes is a super-**common** infection that stays in your body for **life**.
- **Cutaneous manifestations** of HSV infection include:

The commonest presentation of HSV-1:

Pharyngitis and gingivostomatitis, generally seen in children and young adults.

General Features:

Most viral infections show **flu-like symptoms** (Fever, malaise, chills, etc.). In addition, HSV is associated with **difficulty** in chewing and cervical **lymphadenopathy**.

Also, **ulcers** and **exudative lesions** are found on the posterior pharynx and sometimes the tongue, buccal mucosa, and gums.



- HSV disease is usually **not** life-threatening, self-limiting and is more a cause of discomfort than significant morbidity and mortality. However, uncommonly, HSV infections can be **potentially fatal** in certain conditions, which are:

1- Eczema Herpeticum:

- It is an extensive cutaneous vesicular eruption that arises from pre-existing skin disease, usually **Atopic Dermatitis** (eczema).
- Children with AD have a **higher** risk of developing Eczema Herpeticum, in which **HSV-1** is the **most** common pathogen.

Further explanation: HSV is not serious for those with no underlying dermatological problems. Patients with skin diseases (AD) can get critically ill after being infected virally (HSV-1) causing a “combination” of diseases called Eczema Herpeticum.

- Eczema Herpeticum, is a form of **Kaposi varicelliform eruption** caused by viral infection, usually with **HSV**.

Note: Kaposi varicelliform eruption is the name given to a distinct cutaneous eruption caused by some **viruses** (e.g. HSV).



- **Not all** Eczema Herpeticum cases are severe. Only if a **large area** of the skin gets affected with Eczema Herpeticum, it can become **severe**, progressing to **disseminated** infection and **death** if untreated. **Bacterial superinfection** and **bacteremia** via the erupted vesicles are usually the complications that cause mortality.

Note: Superinfection is a **second infection** after an **earlier** different one.

- **Skin eruptions**, in general, are highly **contagious**, so it is important to inform the patient with Eczema Herpeticum to **avoid** contact with others.

*This is a case where the patient was **misdiagnosed** with impetigo during the initial treatment. It turned out that the patient actually suffered from **Eczema Herpeticum**.*

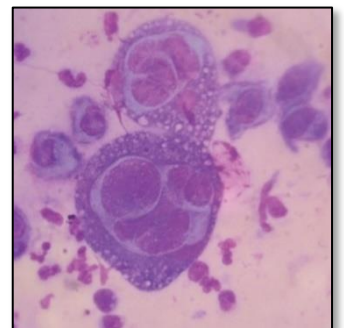


Diagnosis:

- a- It is important to first know the patient's history whether they have/had dermatologic conditions (**AD**).
- b- **Direct fluorescent antibody testing**.
It is **accurate** since it distinguishes between HSV-1, HSV-2, and varicella. Also, it is **rapid** requiring only several hours. However, it is **expensive**.
- c- **Viral culture**.
Its results are **accurate**, reliable and can distinguish between different viruses. Also, it is a **cheap** method. However, results are **slow** requiring 2 days.
- d- **Tzanck Smear**.
It is the **cheapest** method and results are **immediate**, but it requires a **skilled** and experienced observer to interpret the smear.

To perform a Tzanck smear preparation:

Scrapings from the **base** of a **blister** or **erosion** on a glass slide → Staining with **Wright** or **Giemsa stain** → examining under light microscopy for characteristic "Tzanck" cells (**multinucleate keratinocytes**). Tzanck cells can be seen in herpes, varicella, and CMV.



e- **PCR.**

It may be helpful when the cutaneous lesions are **old** or **atypical** and viral particles are **few** in number. However, it may give false positive results.

f- **Pathology lab** can also help in the diagnosis, through the characteristics of the viral cytopathic changes of the epidermis and follicular epithelium present on **H&E** stained tissue.

g- **Immunohistochemistry** (IHC), staining with **antibodies** can also help.

Treatment:

a- **Systemic antiviral** agents should be initiated **as soon as** a diagnosis of Kaposi varicelliform eruption is suspected.

➔ (Oral) **Acyclovir** is the preferred **first-line treatment** in **healthy, immunocompetent** pediatric patients. Usually with a high dose, a 5x/day for 7-10 days.

➔ (IV) **Acyclovir**, for patients with **systemic** involvement or in patients who are **immunocompromised**.

➔ **Foscarnet** is used in patients with **acyclovir-resistant infection** (*last choice*).

***Note:** Foscarnet is **rarely** used (*last choice*), because it causes **nephrotoxicity** in 50% of the patients. But if the patient has a chance of **mortality**, then the risk is worth taking.*

➔ **Valacyclovir** is efficacious and can be dosed **only** twice daily, an advantage over daily multiple dosing of acyclovir. However, it is **cost-prohibitive** (expensive).

b- **Trifluridine and Vidarabine**, are two topical ophthalmic preparations, used for patients with **ophthalmic involvement**. Immediate ophthalmologic consultation is a must for any patient with potential ocular infection.

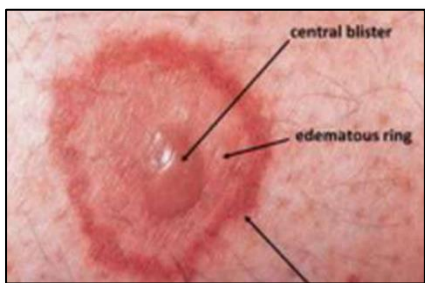
c- **Antibiotics** are used in case of **bacterial superinfection**.

-----End of the 1st medical emergency associated with HSV-----

2- Erythema Multiforme: “2nd medical emergency”

- **HSV** has been associated with up to **75%** of cases of erythema multiforme.
- Erythema multiforme is an **acute, self-limiting** and at times recurring skin condition (rash) thought to occur due to a **hypersensitivity** reaction against certain **infections** (HSV1) and **medications** (antibiotics).
- Erythema multiforme is a term describing **Target lesions** (circular lesions often with a central blister and a **symmetrical** peripheral distribution). It usually shows on **limbs** as a result of **type 4** hypersensitivity reaction, which is a **cell-mediated response**.

*“So Erythema Multiform is actually a rash of target lesions that appear due to **cellular immunity**’s response to **HSV**”*



- Features are often **mouth, genital and eye ulcers** and **fever**, these symptoms are in common with the symptoms of Steven-Johnson Syndrome (SJS).
- **The spectrum of the disease:** *“which is debated, refer to the note”*

Erythema Multiforme minor → Erythema Multiforme major → Steven-Johnson syndrome (SJS) → Toxic Epidermolysis Necrosis (TEN) being the most severe.

Note: Since in Erythema Multiforme there is **minimal** mucous membrane association and <10% epidermal detachment, it is now considered to be a **distinct** condition from **SJS and TEN**. If there is **10%** epidermal detachment this may indicate **SJS**, while if its **>30%** then its **TEN**.

- Erythema Multiforme is frequently seen in **adults** between the ages of **20-40 years**, with **rash** occurring 5-10 days after the onset of viral illness (*flu-like symptoms*). It happens over 3-5 days and persists for 1-2 weeks.
- **Urticaria Multiform** (a benign cutaneous hypersensitivity reaction) is often misdiagnosed as Erythema Multiforme. However, Urticaria Multiform **resolves within hours** (unlike Erythema Multiforme which resolves within weeks).

Causes:

- a- **Idiopathic** (50%), which means with no identifiable cause.
- b- **HSV** (>50%). It occurs 10 days after the acute eruption, or HSV may be the cause even without active lesions.
- c- **Other infections** such as *Mycoplasma pneumoniae*, VZV, HCV, CMV, and HIV.
- d- **Drugs**: Antibiotics and antiepileptic drugs are mostly involved (in SJS as well).
Sulfa drugs, penicillin and ciprofloxacin.

Treatment:

- a- **Identifying** and **removing** the causative agent (e.g. HSV, mycoplasma) or certain medication.
- b- **Supportive care** (pain meds, antihistamines, wound dressing, etc.)
- c- **Antiviral** therapy for HSV causes (oral acyclovir).
- d- Must tell the patient that if the **bullae erupt**, or **systemic symptoms** recur, they **must** return since it can be either SJS or TEN requiring medical emergency since it carries a **mortality** rate.

-----End of the 2nd medical emergency associated with HSV-----

SJS and TEN

“Not related to Erythema Multiforme”

- Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are now believed to be **variants** of the **same condition**, **distinct** from Erythema Multiforme.

Note: TEN is sort of a **severe form** of SJS.

- SJS/TEN are rare, **acute**, serious, life-threatening and potentially **fatal** skin reactions. Complications are very serious and can result in multiple **organ failures**.
- It begins with prodromal symptoms, then progresses to **skin ulcerations** that begin in the **trunk**, **face** and typically involves the **mouth** and **eyes** with **genital sores** as well.
- The patient looks **toxified** (showing cyanosis, poor perfusion, etc.).



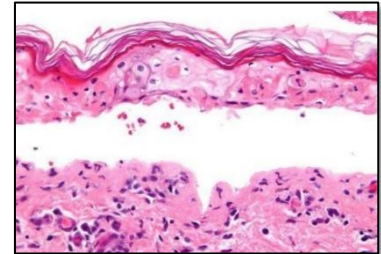
Diagnosis:

- Clinically linked to: **Toxic** looking patient and a positive **Nikolsky sign**.

Note: Nikolsky sign is a method done by **pushing** a bullous over a normal skin with the finger. If the bullous **affects** the healthy skin, this indicates a **positive** Nikolsky sign and a severe activity of a **bullous disease** (e.g. SJS).

- **Biopsy** confirms.

From the biopsy, it shows a **bullous formation** where the epidermis is **detached** from the dermis.



Treatment: Supportive-ICU admission (or burn unit) and some immune-based therapy show promise.

- SJS mortality rate is 3%, while TEN mortality rate is 30% and up to 50%.
- These are the **causes** of SJS and TEN:

1. Drug-Induced Skin Reaction (50% of Stevens Johnson and 90% of Toxic Epidermal Necrolysis)
 1. Highest risk with higher doses and rapid drug introduction
 2. Antibiotics
 1. Trimethoprim-Sulfamethoxazole (**Bactrim**) - most common
 2. Other antibiotics have also been implicated (**Cephalosporins**, **Penicillins**, **Quinolones**)
 3. Anticonvulsants
 1. **Carbamazepine**
 2. **Phenytoin**
 3. **Phenobarbital**
 4. **Valproic Acid**
 4. **Acetaminophen**
 5. **Allopurinol**
 6. **NSAIDs**
 7. **Corticosteroids**
 8. **Vaccinations**
2. Infectious disease
 1. **HIV Infection**
 2. **Herpes Simplex Virus**
 3. **Mycoplasma**
 4. **Hepatitis A**
3. Other causes
 1. Connective tissue disease (e.g. **Systemic Lupus Erythematosus**)
 2. **Pregnancy**
 3. **Radiotherapy**
 4. **Vasculitis**

The doctor asked us to memorize it.

As you can see, the causes are similar to **Erythema Multiforme**. However, in SJS and TEN, the causes are **much more**, and **drugs** are the main causative agents. Unlike in **Erythema Multiforme** where **viruses** are the main causative agents.

After discussing serious skin infections, we will now discuss “normal” infections that are quite common.

Recurrent Herpes Labialis (Oral Herpes)

- The most frequent manifestation of **HSV-1 reactivation**.
- More common in **college students** (37% at 1st yr college and 46% at the 4th yr in the US).
- May be **asymptomatic** or present with symptoms that are **milder** and of **shorter** duration than primary infection (*causing cold sores in about ¼ of college students*).
- Starts as mild **prodromal symptoms** (itch, burn, tingle, etc.), followed by the development of lesions within 48h and then they usually resolve within 5 days.
- **Immunosuppressed** patients may experience **severe mucositis**, with spread to the skin surrounding the mouth.

Note: The **older** and the **more immunocompromised** the patient is, the **more severe** the initial representation of the infection is.

Pathophysiology:

- Caused by **HSV-1** more than HSV-2. However, HSV-2 is on the rise.
- Transmission via **mucous membranes** (kissing), **direct contact** with open skin or **sharing of fomites** (towels, utensils, etc.)
- After 2-20 days of HSV initial contact, symptoms may show. However, shedding is the first 2-4 days maximum.
- After the 1st episode, the virus remains **dormant** in **trigeminal ganglion** and triggers again (recurrence) due to **stressors** such as: Fever, stress, sun exposure, trauma, immune suppression, hormonal changes (menses), fatigue (travel).
- It shows **grouped vesicles** on an **erythematous** base, on the **vermillion border** (*zone between the lip and the adjacent normal skin*). If the patient **scratches** the vesicles, they will **crust** as shown in the 2nd image. Can cause gingivitis, lymphadenopathy, and ulcers on the tongue.



- **Symptoms of the first episode** are usually **severe** with **fever**, **lymphadenopathy**, mouth or gingival **ulcers**.
- **Symptoms of the secondary episode** (after recurrence), lesions that itch, burn, tingle in the first 12-36 hours, then vesicles erupt. It resolves in 7-14 days.

Treatment: We can only **reduce** the symptoms' **duration**, it does **not** remove the virus completely.

- a- **The first episode** → **Acyclovir**, reduces the lesions' time to 4 instead of 10 days and the shedding to only 1 instead of 5 days.
- b- **Recurrence** → **Oral Acyclovir**, reduces eruption healing time by 2 days. **Famciclovir** or **Valacyclovir** can be used too.
- c- **Topical treatment** reduces healing time by one-day maximum.

Genital herpes

- Caused by **HSV-2** (90%) and to a lesser extent HSV-1. It is very similar to **Oral** herpes except that genital Herpes affects the **genital** area and can be **sexually transmitted**.
- It is the **most common** cause of **genital ulcers** (60-70% of STD ulcers).
- It has a high prevalence in **western countries** (12% roughly) or 10-30% of sexually active people. 300k cases show a year in the US alone.
- It is **asymptomatic** in the majority of patients (>2/3). The virus sheds **asymptotically**, so without outbreak, the virus can be shedding in 10-20% of days.

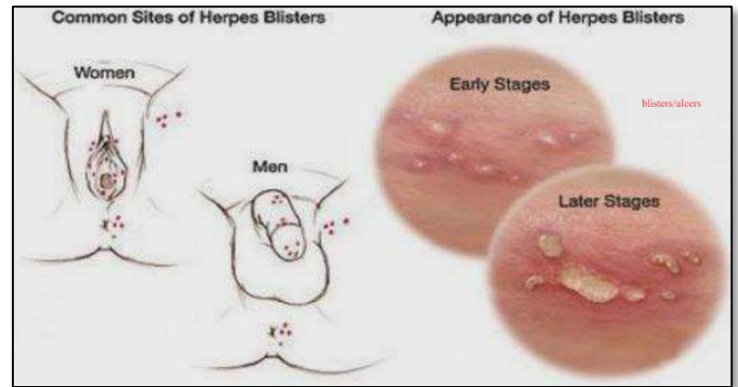
Primary infection:

- May be associated with **fever**, **malaise**, and **adenopathy**.
- HSV's DNA migrates up the infected axon to the affected spinal cord **sensory Ganglion** and persists there **life long**, dormant until the next outbreak.

Secondary infection (recurrence):

- On periodic reactivation, HSV's DNA **migrates down** the axon and erupts again.
- The first infection is the **worst**, then subsequent outbreaks are typically **less severe**.

- **Vesicular eruption:** It is similar to oral herpes, preceded by tingling, itch and burning lesions then vesicles erupt. **Primary vesicles** remain up to **2 weeks**, while **reactivation vesicles** remain for **6-12 days**.
- HSV-1 genital herpes has **milder and fewer outbreaks** than HSV-2.
- Lesions are seen around the **genital area** and the **anal area**.
- In the **early stages** (*more common clinically*) **normal vesicles** are seen while in the **later stages** of the disease they seem to be **crusted** as vesicles erupt and some bacteria may enter.



Diagnosis:

- a- Clinical pictures (symptoms and signs) are diagnostic.
- b- HSV PCR test.

Treatment:

- **Antivirals** for **primary** and **recurrence**. They do not cure but **shorten** the span of illness.
- In pregnancy abstinence, **vaccine** (experimental) + **antivirals** are used.
- As for **measures** for active lesions: loose clothing, ice pack or baking soda compression are used. Active lesions during **pregnancy** may require a **C-section**.
- **Topical antivirals** and low dose **anesthetics** can be given.

Genital Herpes during pregnancy:

- It can be transmitted **vertically** and cause:
 - 1- **Neonatal HSV infections** → Encephalitis or disseminated HSV infection in newborn.
 - 2- **Congenital HSV infection** → Microcephalus, hydrocephalus or chorioretinitis.
-

Other Herpes-Caused Diseases

1- **Herpes gladiatorum**: It is a **mucocutaneous** infection of surfaces such as chest, ears, face, and hands seen in **rugby players** and **wrestlers**.

2- Herpetic whitlow:

- HSV infection of the **finger** which may result from:
 - a- **Auto-inoculation** (existing oral or genital infection).
 - b- **Direct inoculation** from some other environmental source.
- Presents with **vesicles/ulcers** with or without **regional lymphadenopathy**.



Diagnosis: Tzanck smear can be done

Treatment:

- a- Prevent transmission (using a bandage).
- b- **Antivirals** in case of recurrent infection or immunocompromised.
- **Herpetic whitlow** (viral) is different from **Acute paronychia** (bacterial).

Paronychia

It is not a viral infection, but it is discussed to differentiate it from Herpetic Whitlow.

- Paronychia occurs due to **penetrating trauma** (finger biting, manicure, etc.) where **bacteria** from skin (*S. aureus*) or from the mouth (*S. pyogenes* or *Bacteroides*) enter the tissue causing **infection**.
- It is usually **more common** in females since finger biting is more amongst females.
- Infection occurs 2-5 days after trauma, with **pain** at the site and **local inflammation** signs (red, hot, painful and tender).

- Can progress to **abscess formation** and **nail bed infection** (serious), this requires **systemic treatment**.
- Most differentiating feature from Whitlow is that in Paronychia the finger is **more swollen, erythematous** and **painful** with **abscess formation**.



Treatment:

- a- Soak for 15 minutes in **warm water** or **acetic acid** soak (1:1).
- b- If there is **abscess formation** → **Incision and drainage (I&D)**.
- c- **Topical antibiotic** for **mild cases**, e.g. Bacitracin, gentamicin, fluoroquinolone.
- d- **Topical steroid** to **reduce inflammation** (quicker healing).
- e- **Systemic antibiotics** for **prolonged cases** (suspect cellulitis or ingrown nail), must perform (I&D) and then give Cephalexin or Dicloxacillin as first-line therapy.
- f- As a **second line therapy**: **TMP-SFX** or **doxycycline**, especially if you suspect MRSA. *Remember MRSA colonizes the nose, so biting can bring it easily to nail bed.*

-----End of HSV infections-----

Now we will discuss the 2nd virus associated with skin infections, the 1st being HSV.

B- Varicella-zoster virus (Human herpes virus 3).

- It causes **Chickenpox** where 90% of cases occur in **children** under 13 years of age. After that, the virus becomes dormant and its **reactivation** causes **Shingles** (Zoster).
- **Peak onset ages** are 5-9 years old, while the **peak outbreak** time is January to May (cold season-fall).
- Incubation is 10–14 days. Following incubation, the patient may have a 1-2 days **febrile prodrome** (an early symptom of a fever) before the onset of constitutional symptoms of **malaise, itch, and anorexia**. Then **pruritic** (itchy) **red papules** and **vesicles rash** arise (<5mm across).

- Vesicles quickly **pustulate** and form **scabs**, which falls off 1-2 weeks after infection. They appear in **successive crops** 2–4 days, starting on the trunk and face and spreading **centripetally**. *“This means the patient will have **different stages** of vesicles all over and **not** uniformly shaped vesicle”*



- **Dermatomal distribution** of chickenpox: Centripetal.
- Generalized LAP (lymphadenopathy) is common.

Transmission:

- a- **Respiratory droplets**.
- b- Through **direct contact**, where 90% is transmitted by direct contact in households.
- Can also be transmitted **vertically** (transplacental).

Complications of chickenpox:

- May rarely involve the **mucosa** of the **oropharynx** and **vagina**. Other complications include secondary bacterial infection, pneumonitis, and encephalitis.
- The disease may be **severe** in **pregnancy** and the **immunocompromised**.
- If the virus affects a **cranial nerve**, it will become a serious problem.

Treatment:

- Usually, **no** antivirals used. Some guidelines depict oral antivirals in **large** households and in **immunocompromised** patients.
 - **Reducing** the itch through **calamine lotion**, **oatmeal bath** and giving **antihistamine** at bedtime.
 - **Bacitracin** for bacterial superinfection (impetigo) especially for crusted open lesions.
-

Shingles

- In Arabic, it is called fire belt, while in older medicine it is called a flame snake.
- Shingles, or Herpes Zoster, is a **localized recurrence** of **varicella virus**.
- **Dermatomal distribution**: It causes a unilateral vesicular eruption (band-like).

Note: *Shingles* (zoster) are **very painful** and differ in their **dermatomal distribution** (band-like) in comparison to **chickenpox** (centripetal).

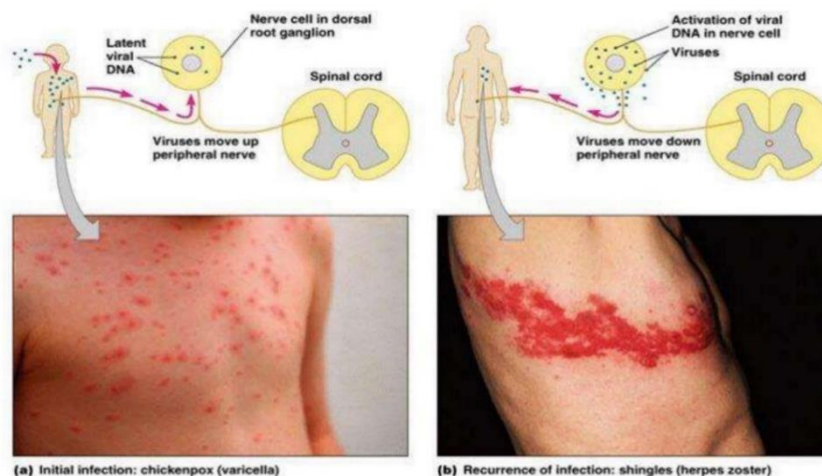
- **Thoracic** and **lumbar** dermatomes are the most common site of infections, **along with** the cutaneous spinal nerve distribution. It is often preceded by 2–3 days of pain in the affected area.

Note: A **dermatome** is an area of skin that is mainly supplied by a **single spinal nerve**.

- In the US, 1 million case incidences show yearly.
- Shingles often occur when a person's immune system is **impaired**. A weakened immune system can be part of the **aging** process; thus, the peak age is in **older** patients (50-80yrs).
- **Women**, particularly during the **menopause transition**, have higher rates of shingles than men (60% are females), most likely due to **hormonal changes** to their immune response.

Pathophysiology:

- The virus infects **dorsal root** ganglionic cells and remains **dormant**, typically following a Varicella zoster infection (**chicken pox**). Reactivation occurs in states of **reduced cell immunity** (older age, immunosuppression and sometimes stress) causing **shingles**.



- It is **highly contagious**. Avoidance of contact is a **must** until rash heals (days to few weeks).
- Most commonly, preceding rash, fever, headache and numbing **along** the affected nerve root. The rash starts **erythematous**, **maculopapular** then clear distinct vesicles erupt. Vesicles turn **cloudy** after 3-5 days and crust by 10 days and may leave a residual **scar**.
- **Lymphadenopathy** with **tenderness** is also a common finding.
- Resolution may take 2–4 weeks.

Diagnosis:

- A rash develops 2-3 days after first symptoms appear and last for 2-4 weeks.
- It follows a dermatomal distribution, proximal then distal to the dermatome.
- Most common sites: Back T1, T2, and face.
- The rash is described as erythematous maculopapular with clear vesicles that crust later.
- **PCR** is the most sensitive and specific test to be done.

Treatment:

- **Antivirals** (*within 3 days of onset is best*) for patients less than 50 years old with more than 50 lesions or complication (*e.g. facial, ophthalmic*).
- **Pain management** with **NSAIDs** and **Opioids**. If the pain persists, **amitriptyline** or **gabapentin** are used.
- **Steroids**.

Variants of shingles

- 1- **Zoster Sine Herpete**: Zoster **without** rash, this is **uncommon**, the pain, prodrome, and fever are all present with no (or little) rash seen. Here **PCR** can be very useful to diagnose it.
- 2- **Ramsay Hunt syndrome** (VZV of the facial nerve): **Rapid** onset with **facial pain**. If the cranial **nerve 8** is involved → **Tinnitus** (ringing in the ears) and **vertigo** (dizziness).

It is seen as a **unilateral herpetic** rash of **ear pinna** with a possibility of hearing loss. It causes **peripheral facial paralysis** (Bell's palsy). “*check the figure in the next page*”

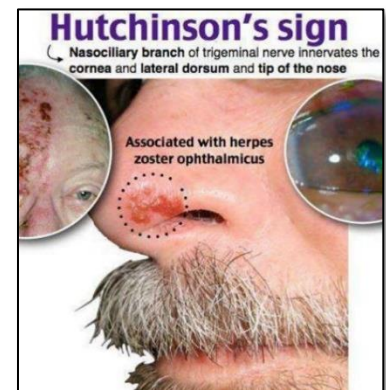
Management: Antivirals, corticosteroids, and painkillers.

*In this case, some doctors might brush this off thinking of this older patient as an old **stroke patient** and send them home. However, by looking at the **ear** you can see the **rash** indicating a **Ramsay hunt syndrome**.*



3- Herpes Ophthalmicus:

- It occurs when the virus is **dormant** in the **trigeminal** nerve ganglion.
- It reactivates more in **advanced age** and **immunocompromised** patients (HIV, cancer, chemo or radiotherapy). It may also reactivate in **systemic illness**, or **stress** (much like typical shingles).
- **Hutchinson's sign** is where vesicles on the **tip** of the nose, or vesicles on the side of the nose, precedes the development of ophthalmicus herpes zoster. It **increases** the risk of ocular involvement by 2 folds.
- **Eye complications:** Keratitis, iritis, episcleritis, and visual loss.
- Recall the **HSV-1** (mainly) also stays dormant in the **trigeminal** nerve ganglion where its reactivation manifests as **Herpes labialis**. Here, the **zoster** virus is dormant in the trigeminal nerve ganglion but its reactivation manifests as **Herpes Ophthalmicus**.



Treatment:

- Consult the ophthalmologist.
- **Antivirals:**
 - a- **Acyclovir**, 800 mg orally 5 times a day for 7-10 days.
 - b- **Valacyclovir**, 1000mg 3 times a day for 1-2 weeks.
 - c- **Famciclovir**, 500 mg 3 times a day for 7 days.
- **Anti-staphylococcal antibiotics.**
- **Corticosteroids**, only under the ophthalmology consultation, since they have a risk of corneal perforation.

Kaposi Sarcoma (HHV8)

- Form of **Cancer**, due to HHV8.
- It has **no** specific dermatomal distribution. If seen, must suspect an **immunocompromised** state, (e.g. HIV).
- Management is aimed at the **cause** of the immunocompromised state.



Few Notes

- **Dermatological Distributions:**
 - 1- **Chickenpox** → Centripetal
 - 2- **Shingles** → Band-like, proximal and distal to the affected dermatome.
 - 3- **Ramsay Hunt Syndrome** → Unilateral rash of ear pinna.
 - 4- **Herpes Ophthalmicus** → Hutchinson's sign "rash on the tip of the nose"
- **Dormancy and Reactivation:**
 - 1- **HSV becomes dormant in:**
 - a- **Cranial nerve ganglion** (trigeminal) → Reactivates as Oral Herpes.
 - b- **Sensory nerve ganglion** → Reactivates as Genital Herpes.
 - 2- **VZV becomes dormant in:**
 - a- **Dorsal root ganglia** (mainly Thoracic and Lumber nerves) → Reactivates as Shingles.
 - b- **Cranial Nerves:**
 - Facial nerve** → Ramsay Hunt Syndrome (hearing loss, vertigo, etc.).
 - Trigeminal nerve** → Herpes Ophthalmicus (Keratitis, iritis, visual loss, etc.).

Some information is prone to updates. I will also inform you of what is not for memorization once I contact the doctor, so stay alert.

Good Luck ♥