MSS

Musculoskeletal System

Doctor 2019 | Medicine | JU

2

Microbiology

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To start with, our subject today will be about skin lesions, particularly vesicular rashes. Knowing the type of rash helps in determining the causative agent of infection, whether it's of ***bacterial***, ***fungal*** or ***viral*** cause. The *majority* of ***vesicular rashes*** are caused by viral agents, *except* a few rare ones, so whenever you see a patient with vesicular rashes in any stage of the disease, you should confine your thinking to the infectious agents seen in the table below. We will be discussing each type in details, so after a couple of lectures you are supposed to be familiar with this table. What helps us in the diagnosis is knowing the age, occupation and lifestyle of the patient, if he was a child with vesicles, then we think about

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	*Rickettsia akari*
Blistering distal dactylitis	*Staphylococcus aureus* or *Streptococcus pyogenes*

chickenpox for example, if he was adult faced exhausting travelling and stress, we think about zoster, if he was a boxer then we think about herpes gladiatorum, if he was a child sitting in a crowded room with other children we think of hand-foot-and-mouth disease.

❖ Viral skin infections/lesions:-

1. **Herpes simplex virus (HSV)**:

The common manifestation for HSV is as a ***cutaneous systemic infection***, which includes:

✓ Pharyngitis (sore throat) with/without gingivostomatitis, the most common presentation of primary HSV-1 infection(**VERYIMPORTANT**)

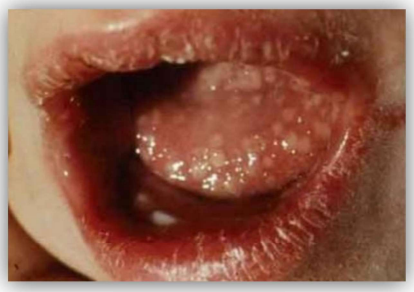
Herpes vesicles do not have to be existed at the beginning

- ✓ Constitutional symptoms, such as fever, malaise, fatigue & myalgia
- ✓ Difficulty chewing and cervical lymphadenopathy

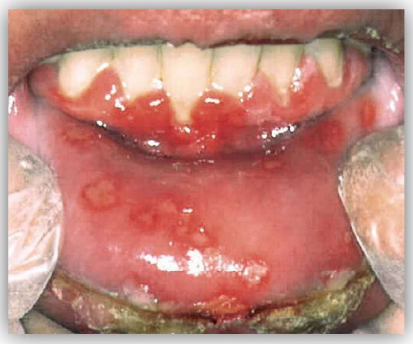
Notice the following:-

- You can't diagnose a patient based on constitutional symptoms, but they might give you an idea that there is a systemic viral load
- Malaise: Feeling of discomfort / Myalgia: Muscle pain
- Lymphadenopathy means enlargement of the lymph nodes in the cervical area, according to the inflammatory response with CD8+ T cells due to having herpes in the body

- ✓ Ulcers and exudative lesions are found on the *posterior pharynx* and sometimes the tongue, buccal mucosa and gums

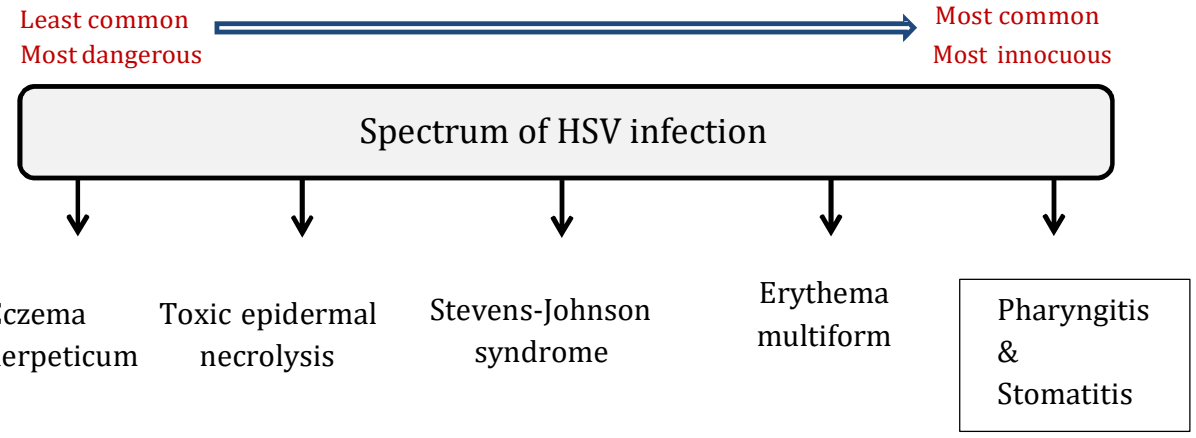


Pharyngitis



Stomatitis

Note: These eruptions resemble the vesicles that are seen in the skin, but they aren't elevated in the mucosal membranes because of the absence of epidermis (keratin), thus, they will ulcerate.



I. **Eczema herpeticum (form of Kaposi varicelliform eruption)**:

It is an extensive cutaneous vesicular eruption caused by HSV-1 that arises from ***pre-existing skin disease***, usually atopic dermatitis, which is also known as eczema.

Children with atopic dermatitis (eczema) have a **higher risk** of developing eczema herpeticum.

It can be severe, progressing to *disseminated infection* targeting every organ system and if untreated, it might lead to death.

Bacterial super-infection and *bacteremia* are usually the complications that cause mortality.

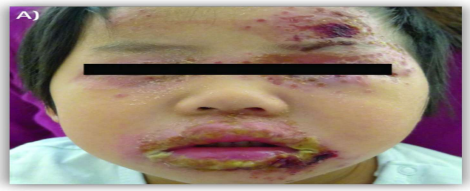
For further clarification:

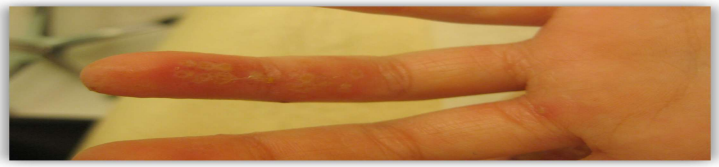
HSV isn't serious for those with no underlying dermatological diseases. On the other hand, patients with skin diseases can get seriously ill after being infected virally with HSV-1, which will facilitate the passage for another bacterial infection (***bacterial on top of viral***), thus producinga ***systemic wide infection***

that requires a ***systemic therapy***.

Clinical case: (*Look at the picture on the left while reading*)

A 9 years old girl with a history of atopic dermatitis with recurrent skin infections presented to the emergency department suffering from skin disease, fever, fatigue, bronchial asthma and malaise. She was diagnosed as impetigo and given antibiotics as treatment. After a while, she came back to the hospital claiming that she didn't feel better. On physical examination, the physicians noticed vesicular eruptions on her hand. What do you think is the proper diagnosis?





This picture is about a person put his finger in his affected mouth with herpes, it is not an Eczema

Answer: Eczema herpiticum, why?

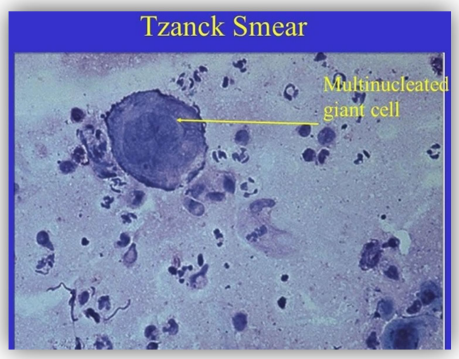
Because it doesn't make sense for impetigo, a superficial bacterial skin infection, alone to cause all of these symptoms (fever, fatigue …), thus we must be sure that she is also infected with other infectious agents. Since on physical examination doctors found vesicular eruptions and approximately ***vesicular = viral cause***, thus in order to solve this case, we must confine our thinking to the agents found in the table and search for a disease with ***bacterial on top of viral infection***.

Diagnosis: (we need a medical approach to investigate having HSV if symptoms were not just malaise or cold, or if the patient was immunocompromised, so we need to know whether this is HSV or not)

- ✓ ***Direct-fluorescent antibody testing (antigen-antibody rxn)***
- ✓ *It is performed through adding antibodies to the sample, if they are bound, then the sample has this virus.*
- ✓ Pros: Specific (distinguishes HSV-1 from HSV-2) and rapid (hours)
  Cons: Expensive
- ✓ ***Viral culture (requires host cells for each specific virus)***
- ✓ *It is performed through having cells that a virus attacks, we add our sample to these cells, if they are attacked and cleared, then the sample contains the virus.*
  Pros: Specific (accurate), reliable and cheap
  Cons: Slow (2 days)
- ✓ ***Tzanck smear***
  Pros: Immediate results and cheap
  Cons: Non-specific (can't distinguish between viruses) and it's very user-dependent (requires skilled and experienced observer)

How do you prepare a Tzanck smear?

- Using a blade, gently deroof and scrape the base of the lesion
- Smear the tissue onto a clean microscope glass slide
- Allow the specimen to dry in the air then fix it with fixatives
- Stain the specimen with ***Wright/Giemsa stain***
- Under the light microscope, look for Tzanck cells ***(multinucleated giant cells)***, which is very common immunological reaction for viral infections



- ✓ ***PCR***
  Extract DNA from all cells then look for specific genes that are only present in specific viruses. Extracting DNA takes time, so it's a slow process but very effective and not that expensive
- ✓ ***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.
- ✓ ***Immunohistochemistry*** (staining with antibodies) can also help

Treatment:

Treatment with ***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 otherwise healthy (*immunocompetent*) pediatric patients. Usually given at *high doses*, 5x/day for 7-10 days
- ✓ **IV acyclovir** is for patients with *systemic involvement* or in patients who are *immunocompromised*
- ✓ **Valacyclovir** is efficacious and *can be dosed only twice daily*, an advantage over daily multiple dosing of acyclovir. However, it is *cost-prohibitive* for some patients
- ✓ **Foscarnet** should be used in patients with *severe cases, acyclovir-resistant infection*. This is an *antiviral medication* that works on herpes viruses; however it causes *nephrotoxicity in 50% of patients*, but when it comes to mortality, you take morbidity over mortality
- ✓ Two topical ophthalmic preparations, **trifluridine** and **vidarabine**, are available for *use in patients with ophthalmic involvement*

II. Erythema multiforme:

It is an *acute*, not gradual or insidious, *self-limiting* and *recurring (happen repeatedly)* skin condition that thought to occur due to *type IV hypersensitivity reaction* against certain infections, intracellular infections, including HSV-1, and medications (antibiotics and antiepileptic).

The *majority* of cases (75%) are caused by *primary HSV-1 infection*,

but thereare other causes, including bacterial infections.

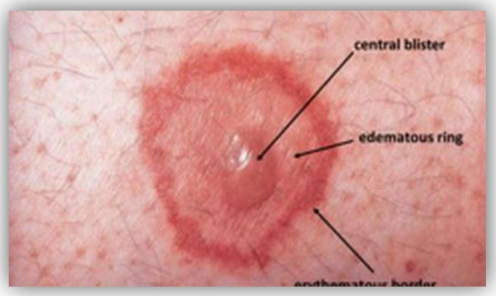
Erythema multiforme is a term describing *target lesions*, which are circular lesions with central blisters and well-defined margins.

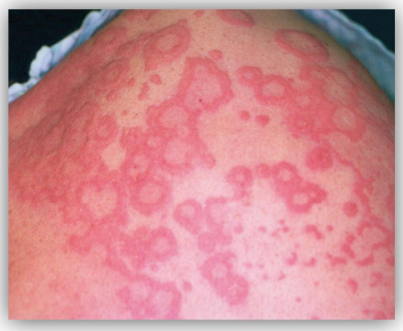
Not all patients with HSV infection will get erythema multiform; this is because of the difference in mechanism of which the immune system of different patients reacts towards delayed hypersensitivity reactions for these kinds of viruses.

It's less dangerous than eczema herpeticum, with approximately no mortality rate. It's not considered a serious condition; it can be treated in the clinic.

With its minimal mucous membrane association and <10% epidermal detachment, *erythema multiforme is now considered to be a distinct condition from SJS and TEN*, i.e. ***it can't progress to SJS and TEN***

Frequently seen in adults between the ages of 20-40 years, with rash occurring 5-10 days *after the onset of viral illness*, happen over 3-5 days and persist for 1-2 weeks. Urticaria is the major differential here, which resolves in hours.





Now, if the rash took so long and it *involves mucous membranes* of the mouth, nose and genitals, also producing *eye ulcers and fever*, it's not erythema multiform, it's ***Steven-Johnson syndrome (SJS)***.

It's more severe than erythema multiform, with 3% mortality rate. It can't be treated in the clinic, because it needs clinical supervision.

SJS is probably caused from administration of *antimicrobials*, *anticonvulsants* (antiepileptics) or other medications.

When taking the history of the patient that was taking antimicrobials,

for example, and he was diagnosed with SJS, you must change the drug because it's most probably what caused the allergy (type IV hypersensitivity reaction)

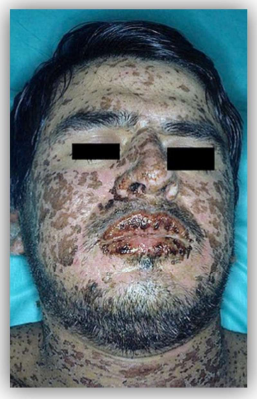
SJS = erythema multiform all over the skin + mucous membranes involvement

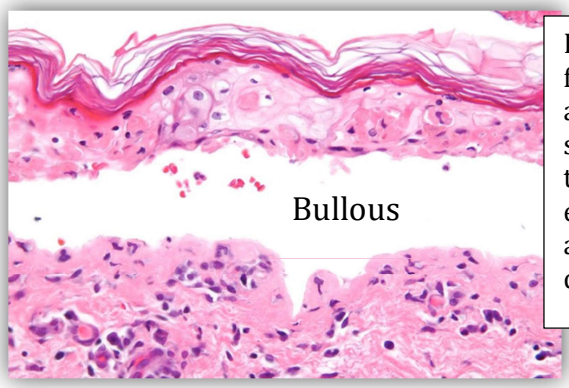
Now, if it involves mucous membranes with toxic looking patients and bullous formation (positive nikolsky sign), *Steven-Johnson syndrome* can progress to ***Toxic epidermal necrolysis (TEN)***.

TEN is more severe than SJS, with 30% mortality rate. It requires ICU admission and in some cases immune based therapy.

It begins with prodromal symptoms, then progress to *skin ulcerations* affecting the entirety of dermis that begin in trunk and face and typically involve the mouth, the eyes and with genital sores as well.

Diagnosis of TEN: Clinical is enough, biopsy confirms





Bullous formation is about separating the epidermis and the dermis

Biopsy indicating bullous formation

TEN = SJS + toxic looking patient with bullous formation

Causes of erythema multiform, SJS & TEN:

- ✓ HSV (> 50%), might cause infection even without active lesions *it occurs 10 days after acute eruption
- ✓ Other infections (***Mycoplasma pneumoniae***, VZV, HCV, CMV & HIV)
- ✓ Drugs →antibiotics and antiepileptic drugs are mostly implicated (in SJS), sulfa drugs, penicillin and ciprofloxacin. However, other medications may cause these diseases
- ✓ Idiopathic (50%) (they give the patient cortisol to reduce the inflammatory response)

Treatment of erythema multiform, SJS & TEN:

- ✓ Remove the precipitating factor:
  The primary treatment of allergy is to ***remove the allergen***, whether it's HSV, Mycoplasma or medication
- ✓ ***Supportive care*** (pain meds, antihistamines, wound dressing)
- ✓ Antiviral therapy for HSV causes – ***Oral acyclovir***

Extra Treatment for TEN patients:

- ✓ ICU/Burn unit admission and immune based therapy
- ✓ Patients must return if bullae erupt or systemic symptoms recur

**patients with Erythema multiforme are treated in the clinic, SJS patients are treated in the ward, TEN patients in the ICU

Now, let us discuss the cutaneous forms of viral infections:

- ➢ labialis and genitalis caused by HSV
- ➢ Chickenpox and shingles caused VZV

Recurrent herpes labialis:

- Most frequent manifestation of HSV-2 reactivation
- Most common in college students in the west (37% of year1 students, 46% of year4)

- May be *asymptomatic while the viral is shedding* or present with symptoms that are milder and of shorter duration than primary infection (cold sores are in about the quarter of students) (same in genitalis as well)
- Mild prodromal tingling is followed by the development of lesions within 48h and usually resolve within 5 days.
- Immunosuppressed patients may experience ***severe mucositis***, with spread to skin surrounding the mouth (the picture of affected finger in page 4)

Pathophysiology of labialis:

- HSV-1 > HSV-2 (In genitalis, HSV-2 > HSV-1)
- Transmission via mucous membranes (kissing), direct contact with open skin or sharing of fomites (towels, utensils, …etc)
- Shedding occurs in the ***first 2-4 days*** maximum
- Symptoms (primary vesicular eruptions) start to appear ***2-20 days*** after contact and direct inoculation
- First episode is more severe (fever , LAP, mouth or gingival ulcers) than other recurring episodes
- After the episode, herpes 1, 2 & 3 ***always remains dormant*** in the nearest sensory ganglion, that's why herpes is a ***life-long infection***
- ***Secondary episode , after recurrence, lesions (itch, burn, tingling first 12-36 hours, then vesicles erupt…heal in 7-14 days***

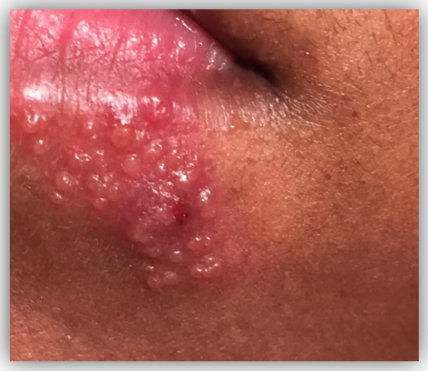
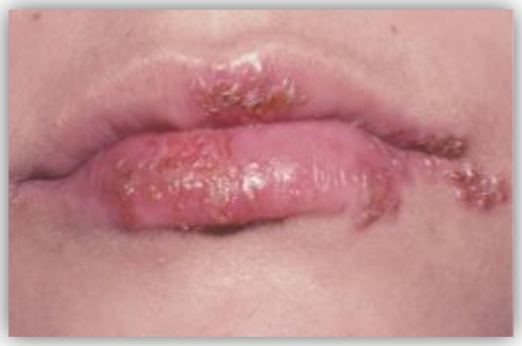
Note: Dormant viruses can't be treated with antivirals, because almost all antivirals target specific metabolic reaction in the virus, which isn't taking place in a dormant virus

- Stress triggers the recurrence of dormant virus, examples of stressors include: fever, sun exposure, trauma, immune suppression, hormonal changes (menses) & ***travelling*** (***fatigue***)

Note: Stress here doesn't mean anxiety, it's any factor that threat your immune system

So, viruses always check your immune system:

- If it's competent → Virus remains dormant
- If it's compromised → Recurrence of virus takes place

These two figures show how labialis looks, however, genitals and shingles also have the same appearance

HSV-1is more common around the mouth, while HSV-2 is more common in the genitalia
both can be transmitted sexully, HSV-2 is rising in terms of being around the mouth

Treatment:

Treated symptomatically to reduce the symptom duration and the viral shedding, but we *can't cure the disease (no removal of virus)*

- On first episode give ***acyclovir***, which reduces the lesion time from 10 days to 4 days and viral shedding from 5 days to only 1 day (less shedding = less spread).
- On Recurrence:
  - ✓ Oral: ***acyclovir***, reduces eruption healing time by 2 days, ***famciclovir*** or ***valacyclovir***
  - ✓ Topical, reduces healing time by less than 1 day

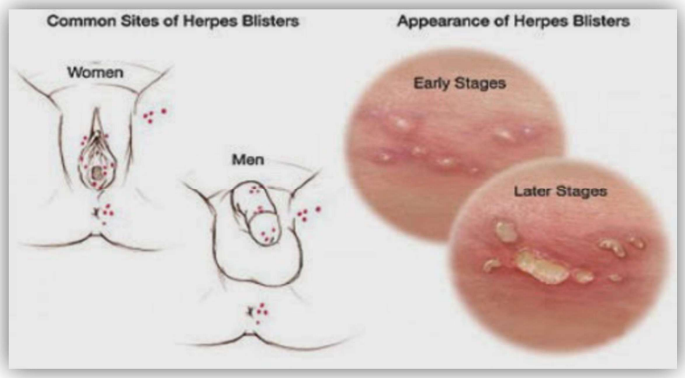
Genital herpes:

- Caused by HSV-2 (90%) and HSV-1(although now HSV-1 on the rise!)

  High prevalence in western countries (12% roughly) or 10-30% of sexually active people.
- The most common cause of genital ulcers (60-70% of STD ulcers)
- 300k cases a year in US alone!
- Asymptomatic in majority of patients (>2/3)→ (helps in spreading)

- Virus sheds asymptomatically
- • Virus SHEDS ASYMPTOMATICALLY (so without outbreak the virus can be shedding in 10-20% OF DAYS!) Primary infection • (Primary Genital Herpes) may be associated with fever, malaise and adenopathy • HSV DNA migrates up the infected axon to the affected spinal cord sensory Ganglion (similar story in the herpes viruses)
- HSV persists in the sensory Ganglion ***life-long***, dormant until next outbreak
- • On periodic reactivation, HSV DNA migrates down axon and erupts again
- First infection is worst, then subsequent outbreaks are typically less severe
- Vesicular eruptions take place also, - Similar to oral herpes, preceded by tingling, itch and burning
- *primary vesicles remain up to twoweeks*, *reactivation 6-12 days*
- HSV-1 genital herpes is milder and fewer outbreaks than HSV-2

The figure shows common sites of blisters and their 2 forms. ***Early form*** present as ***clear vesicular form*** and the ***late form*** present as ***a cloudy vesicle*** that looks like it will turn into pustule but it's still a vesicle.



Diagnosis:

- Clinical picture alone is helpful. By looking at the color of the vesicles you can tell if it's an early or late stage.
- PCR test for HSV confirms

Treatment:

- Antivirals for primary and recurrence, they shorten the span of

illness but they don't cure the disease (no removal of the virus)

- DURING PREGNANCY, might transmit vertically and cause neonatal HSV infection (can cause encephalitis or disseminated HSV infection in new born, or cause congenital(the virus has ascended while the baby is forming not while the baby is going through the birth canal) HSV infection which may lead to microcephalus or hydrocephalus or chorioretinitis)
- In pregnancy abstinence, vaccine (experimental) and antivirals
- If the female has *active lesions* by the time of delivery, the baby might contract herpes encephalitis from his mother, so we should
- give her vaccines and antivirals during her pregnancy period.

If the *active lesions* still present, *cesarean section* is a must!

Note: Active lesions of labialis in pregnant women don't infect babies during delivery.

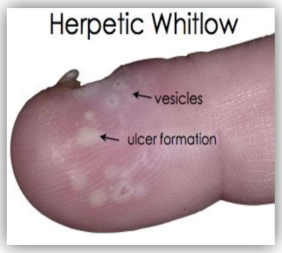
- As for measures for active lesions: loose clothing, ice pack or baking soda compression might help
- • Topical antivirals and low dose anesthetics can be given

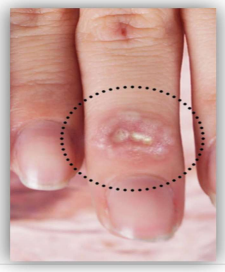
Herpetic whitlow:

- HSV infection of the finger (*Localized HSV replication*), which may result from auto-inoculation from existing oral/genital infection, or by direct inoculation from some other environmental source.
- It presents with *vesicular eruptions* ± *regional lymphadenopathy*.
- Differentiate from acute paronychia (bacterial)
- Tzanck smear can be done to help in ***diagnosing***.

Treatment:

- Prevent transmission through bandages
- Antivirals, in case of recurrent infection or immunocompromised.
- Dermatologists usually follow cryotherapy treatment, in which they use liquid nitrogen to phase of these parts of the skin, thus preventing viral replication. Topical treatment doesn't help a lot.





Herpes gladiatorum*:*

Mucocutaneous infection of a of surfaces such as chest, ears, face, and hands , who has a very long intimate contact with a patient with herpetic whitlow on his digits,such as rugby players and wrestlers, so the healthy one might be inoculated developing what is known as herpes gladiatorum.

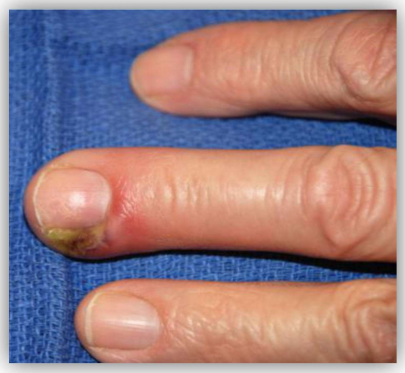
Paronychia:

- Paronychia usually more in females
- Bacterial infection, either from the skin (Staph. aureus) or from the mouth (Streptococcus pyogenes/Bacteroides), due to penetrating trauma (finger biting, manicure ...etc).
- *Infection* usually takes place *2-5 days after trauma*.
- *In contrast to herpetic whitlow*, it presents with pain and local signs of inflammation, such as redness, hotness, swelling & tenderness.
- Can progress to *abscess formation* and *nail bed infection* (severe).

Treatment:

- soak for 15 minutes in warm water or acetic acid soak (1:1)
- • I & D (if abscess)
- *Topical antibiotics in mild cases*, such as bacitracin, gentamicin, fluoroquinolone.
- • Can add topical steroid as well to reduce inflammation (quicker healing)
- • Prolonged case*: Systemic antibiotics in prolonged cases* (cellulitis/ingrown nail)
- • Second line therapy : TMP-SFX or doxycycline-especially if you suspect MRSA • Remember MRSA colonizes nose , so biting can bring it easily to nail bed!

To sum up, anti-staph local medications is all what you need. If they didn't work, you should start to worry; because in this case maybe the nail bed has an abscess, which requires drainage alongside with systemic therapy.



2. **Varicella-zoster virus** (**Human herpesvirus 3**):

There are two manifestations for VZV, which are:

- For primary infection → Chickenpox
- For secondary infection (recurrence) → Shingles

We all know from the introductory course that you can't have shingles, unless you have had chickenpox before. These days with the evolution of vaccines, even if the person is vaccinated against VZV he might have shingles; this is because when he got vaccinated at a very young age, at some point, he had chickenpox with *subclinical manifestations (atypical infection)* that neither he nor his parents remember.

Chickenpox:

- 90% of cases occur in children under 13 years of age. this is in what age you suspect it most SCHOOL)
- Peak onset ages 5 to 9 years old. (January to May- cold season-fall)
- Incubation period is 10-14 days.
- **Transmission**: *Respiratory droplets, direct contact or vertical*
- 90% transmitted by direct contact in house hold (pox parties!)

Following incubation, a patient may have a *1-2 days febrile prodrome* before the onset of constitutional symptoms (malaise, itch, anorexia) and then *papulo vesicular rash* (<5mm across), which is ***itchy***! Pruritic red papules and vesicles

Note: In order to differentiate chickenpox from other diseases, usually it appears in *successive crops over 2-4 days*, *macules* then *papules* then *vesicles* and finally vesicles break-up and become *small crusted lesions*, this sequence of events doesn't happen simultaneously all over the skin, i.e. when you look at the patient you will see *all different stages and forms of chickenpox (macules, papules, …) at the same time*.

This helps in diagnosis

Complications of chickenpox:

- *Itchy rash is the main symptom of chickenpox.*
- Secondary bacterial infection, pneumonitis and encephalitis.
- Disease may be severe in pregnancy (congenital varicella or pneumonia for the mother)and the immunocompromised.
- May rarely involve the mucosa of the oropharynx and vagina.

Treatment:

- Reduce itch by *calamine lotion, oatmeal bath or give antihistamine* at bed time.
- Bacitracin for bacterial super-infection in crusted open lesions
- No need for antiviral Some guidelines depict oral antivirals in house holds that are large and in immunocompromised patients) **(VERY IMPORTANT)**

Shingles: (الحزام الناري)

- Localized recurrence/reactivation of varicella virus.
- Causes a *unilateral vesicular eruption in a dermatomal distribution*.
- • This is typically and often preceded by 2–3 days of pain in the affected area.

Remember: Dermatome distribution is a cutaneous area that is supplied by a single spinal nerve root.

- *Bilateral shingles is much more severe* and was called in older medicine "flame snake" (if the head of the snake eats the tail the patient dies).
- Most commonly happens in *thoracic and lumbar dermatomes* along the cutaneous spinal nerve distribution.
- U.S (300 million population) has a 1 million case incidence yearly.
- Usually happens in elder patients; because of weak immune system, and 60%females; because of hormonal-change (menopause).

Pathophysiology of shingles:

- The virus infects dorsal root ganglionic cells and remains dormant, typically following a varicella-zoster infection (chickenpox).
- Reactivation of the dormant virus causes shingles.
- ***When does reactivation occur?*** In states of reduced cell immunity, older age, immunosuppression and sometimes stress. (Same as reactivation of HSV-1 and HSV-2)
- *It's contagious.* Must avoid contact until rash heals (days to few weeks!)
- Most commonly preceding the rash: fever, headache and numbing along the nerve root is reported.
- The rash starts as *erythematous and maculopapular*, then later on it will appear as *clear distinct vesicular eruption.*
- • Vesicles turn cloudy after 3-5 days and crust by 10 days, may leave residual scar.
- *Pain is the main symptom of shingles, why? Because the nerve where the reactivation takes place gets inflamed and unmyelinated by our immune system after an attempt to stop the viral replication.*
- Resolution may take ***2-4 weeks***, why? Because this is the time needed for the axon of the infected nerve to get re-myelinated again.
- • LAP with tenderness is also a common finding

- Atypical rash might develop (shingles without rash/rash in uncommon site). To make sure it's shingles, make a PCR test.

Diagnosis: (Mostly clinical)

- Rash develops 2-3 days of first symptoms and last for 2-4 weeks
- Follow a dermatomal distribution, proximal then distal to the dermatome
- • Most common sites: Back T1, T2
- • And face (see later)
- Rash is described as *erythematous maculopapular with clear vesicles that crust after a week or so*.
- PCR is the most sensitive and specific test to be done (atypical rash)

Treatment:

- Antivirals within 3 days of onset 50> y/o and more than 50 lesions or complication (facial, ophthalmic); in order to limit viral replication.
- Anti-pain medications:
  Pain management NSAID → Opioids → Refractory pain amitriptyline or gabapentin
- Vitamin B12; helps in re-modeling of the myelin sheath.
- Steroids.

Variants of shingles:-

- ***Zoster Sine Herpete:***

  Zoster without a rash, this is uncommon, pain, prodrome and fever are all present with no (or little) rash seen. (Rare, but on the rise)

- ***Ramsay hunt syndrome:***

  VZV of the facial nerve, rapid onset with facial pain• Tinnitus and vertigo if cranial nerve 8 is involved, peripheral facial paralysis due to reactivation of VZV and sometimes it can affect the earleading to hear loss. • Management (similar to bell's palsy = facial nerve paralysis due to VZV reactivation) : antivirals+corticosteroids+pain killers

➢ ***Herpes Ophthalmicus:***

The virus is dormant in the trigeminal nerve ganglion, reactivates more in advanced age and immunocompromised patients(HIV, cancer, chemo or radio therapy). May also reactivate in systemic illness, or stress (much like typical shingles)

Has a typical sign called Huntchinson's sign, which is a vesicular eruption atthe tip of the nose that most of the time indicates ocular involvement Can cause eye complications (keratitis, iritis, episcleritis) VISUAL LOSS

Treatment: Consult the ophthalmologist

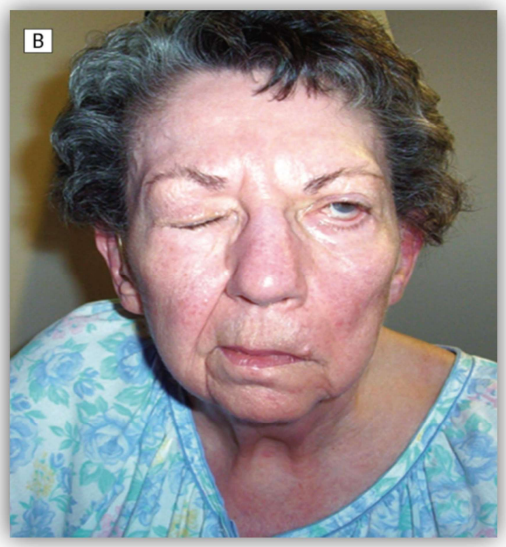
- Antivirals, such as acyclovir(acyclovir 800 mg PO five times a day for 7-10 days), valacyclovir (1000mg 3 times a day for 1-2 weeks) and famciclovir(500 mg 3 times a day for 7 days)
- Anti-staph antibiotics
- Corticosteroids, only under the ophthalmology consultation-- has a risk of corneal perforation).

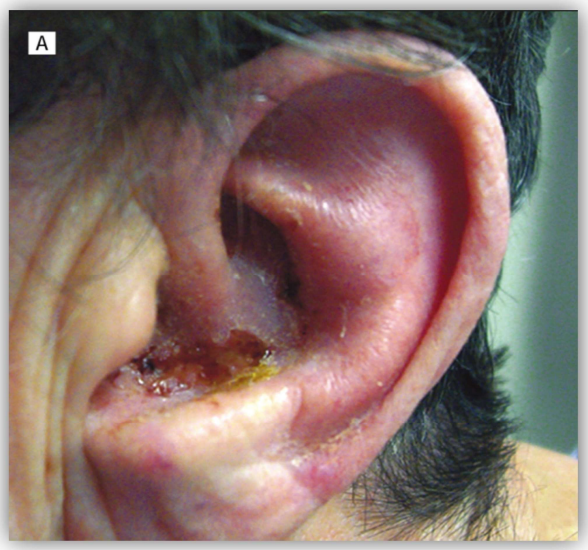
3. **Kaposi sarcoma-associated herpes virus** (**Human herpesvirus 8**):

- Form of a cancer
- Not in a dermatomal distribution
- If seen must suspect immunocompromised state (e.g. HIV)
- Management is aimed at the cause of the immunocompromised

Clinical case: (*Look at the pictures while reading*)

A 78 years old female with a medical history of diabetes, hypertension and stroke presented to the emergency department complaining of pain, hypoacusis, otorrhea, facial asymmetry and left hemiparesis. Due to the suspicion of a new stroke event, she was given medications and limitations for her diet and they wanted to send her back home. Before she leaves, the physician noticed a rash in her left ear, he ordered a PCR test and he found that there is a virus in one of the cranial nerves. What do you think is the proper diagnosis?





Answer: Ramsay hunt syndrome

Keywords → Pain, facial asymmetry, rash in her ear, virus in CN.

THE END