433 Teams

DERMATOLOGY

Lecture

(7)

Common bacterial & viral infections

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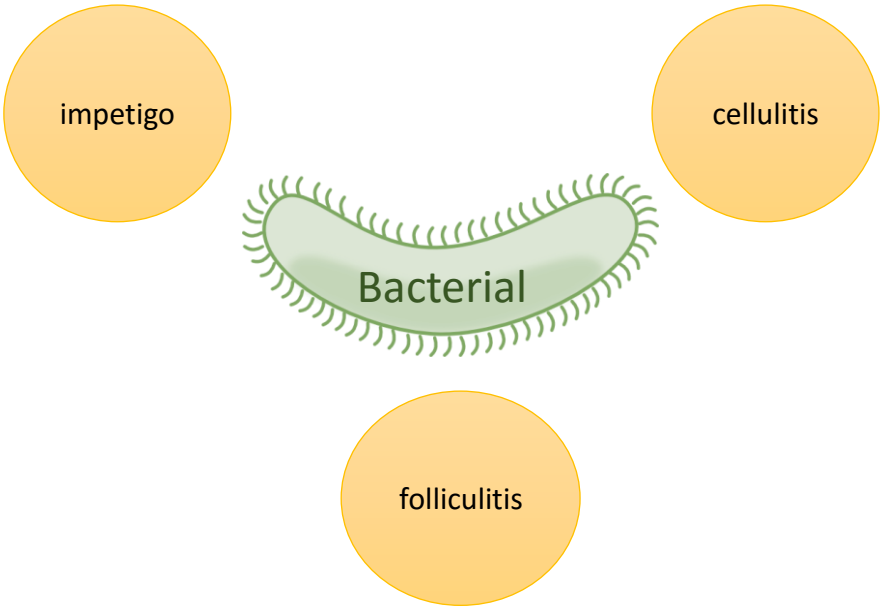
Objectives: Not given

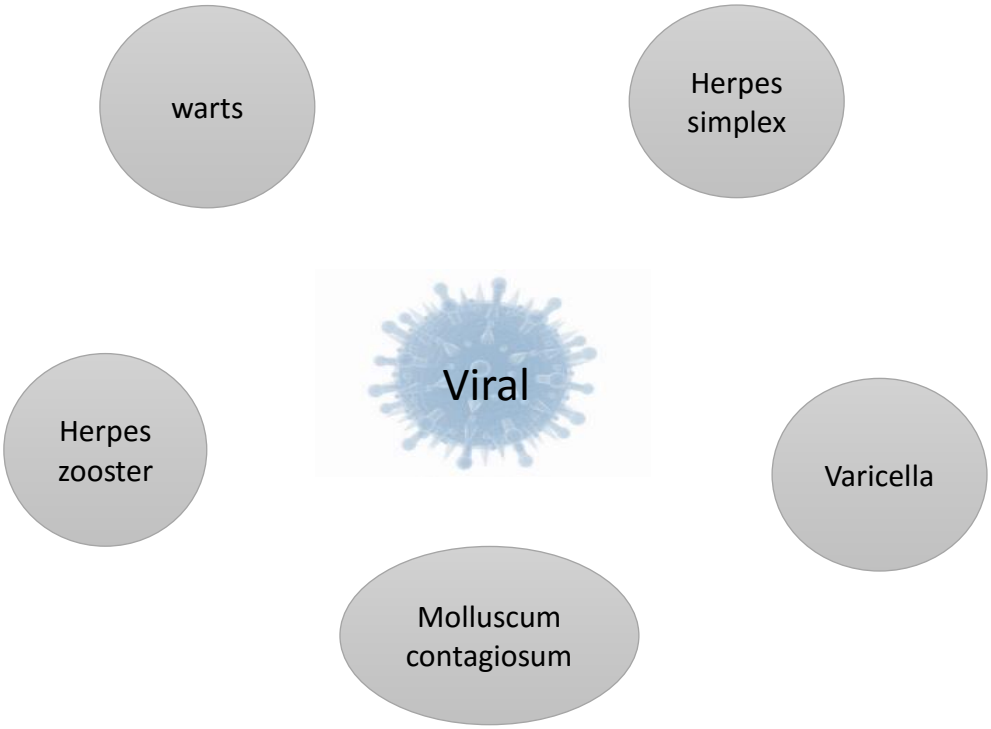
from 432 team:

1. General understanding of the causative organisms of common skin infection(CSI).
2. Focus on CSI clinical presentation.
3. Overview of the basic investigations done and general knowledge of first line therapy.

CSI= common skin infections

Color index:	slides,	doctor notes,	432 notes	important


impetigo
cellulitis
Bacterial
folliculitis


warts
Herpes simplex
Viral
Herpes zooster
Varicella
Molluscum contagiosum

Bacterial infections:

Predisposing factors

- ✓ **Previously damaged skin**
- ✓ **Impaired host immunity**

Normal skin flora:

- Coagulase negative staphylococci (staph. Epidermidus)
- Diphtheroids

Impetigo:

A superficial skin infection

Common in children (2-5 yrs)

Etiology: **Strep pyogen, Staph aureus**

Clinically:

Presents as thin-walled **vesiculopustules** on an erythematous base with yellow gold crust **(honey crust)**

Common sites: face, extremities.

Commonly associated with minor skin trauma.

Systemic symptoms (not usual).

Rarely complicated by APSGN (Acute post-streptococcal glomerulonephritis) (nephritogenic strains).

It could be:

Primary: if there are no previous skin lesions

Secondary: if the infection occurs on top of a previous skin lesion (impetigo on top of eczema or herpes).

Predisposing factors:

- Warm, humid climate
- Poor hygiene
- Trauma
- Insect bites
- Immunosuppression

Investigations:

Bacterial cultures (confirmatory)

Serology (rarely indicated)

Treatment:

Localized disease → **Topical antibiotics** (Bacteroban), and good general hygiene

Extensive lesions / Infection with nephritogenic strains → **Systemic antibiotics** (1st generation cephalosporin) and good general hygiene

Bullous impetigo:

- Is a variant of impetigo caused by staph aureus (Group II phage type 71)
- Clinically presents as:

superficial flaccid vesiculopustules

↓ rupture

spread & coalescence of lesions

↓

rounded denuded areas

- ***Treatment:*** **antistaph systemic antibiotic**

Note: does not cause APSGN because it is caused by staph

Folliculitis:

Is an inflammation of the upper part of the hair follicles that can be infectious or non-infectious.

Infectious folliculitis is commonly caused by S aureus (can also be due to viral, fungal infections or infestation)

- **It could be:**
  - ✓ **Primary:** if there are no previous skin lesions
  - ✓ **Secondary:** if the infection occurs on top of a previous skin lesion (e.g. folliculitis on top of eczema).

Predisposing factors: excessive itching, immune compromised (e.g. diabetics)

Morphological features: multiple small papules / pustule on an erythematous base in follicular distribution areas (scalp, face, trunk, legs)

Course is usually **chronic and relapsing**.

Management:

Investigations: swab for gram stain and culture.

Treatment: Topical or systemic antibiotics directed by culture findings. (some people **carry** pathogenic bacteria as normal flora [mostly **in the nostrils and axilla**] and present as **frequent** folliculitis. they can be treated by **eradication** of the bacteria by applying topical antibiotics)

Furuncle (boil)	Carbuncle
Inflammation of **deep portions** of hair follicle. **Caused by S. aureus.** **Treatment:** drainage and **systemic antibiotics** 	Deeper infection composed of **multiple interconnecting abscesses** usually arising in several contiguous hair follicles. Also **caused by S. aureus.** **Treatment:** excision and systemic antibiotics. 

Read only!!

Folliculitis vs. furuncles

Folliculitis is inflammation of the **upper portion** of the hair follicle can be infectious or non-infectious while furuncles is **S. aureus infection of the deep portion** of the hair follicles. **Furuncles are more erythematous and painful**. Furuncles require drainage and systemic antibiotics while folliculitic can be treated with topical anti-biotics.

Cellulitis:

Is an acute bacterial suppurative inflammation of the skin, particularly the deeper subcutaneous tissues.

Caused by:

- Strep A (GAS) (e.g. strep pyogenes).
- Staph aureus
- H. influenza

Common sites:

Face and lower extremities

Clinical features

preceding wound or trauma

markedly red, tender, warm swelling

can lead to systemic S&S: fever, tachycardia, LAP (lymphadenopathy)

risk factors:

- DM
- Venous stasis
- Immunodeficiency

Complications: lymphadenopathy if recurrent

Investigations: **cultures** of:

- FNA
- Skin biopsy
- Blood

are usually negative

H. influenza cellulitis

- Seen in children <2 yrs old
- Facial and periorbital
- Dusky red or blueish discoloration
- The child may be extremely ill

Treatment:

1. Treat any local skin damage.
2. Appropriate systemic antibiotics
   - Oral for mild cases (e.g. cephalexin)
   - IV or IM for more severe cases

✓ Recurrent cases can be given long term prophylactic antibiotics (e.g. penicillin)

✓ Orbital and periorbital cellulitis require admission and involvement of ophthalmologists.

erysipelas

Read only!!

Cellulitis vs erysipelas

Erysipelas are **superficial** infection of lower dermis while **cellulitis are infections of the subcutaneous tissue**. The clinical difference is that **erysipelas have better defined raised edges** compared to cellulitis. Lymphatic involvement is more pronounced in erysipelas. **Management is similar.**

Viral Infections:

Warts:

Warts (verrucae) are common and benign skin tumors resulting from infection of epithelial cells by human papillomavirus (HPV)

The doctor said non need to memorize the subtypes

HPV 1	deep planter warts
HPV 2,4	common warts
HPV 6,11 acuminatum	condyloma
HPV 16,18,31,33	genital neoplasia

HPV

- Ds-DNA virus (papovavirus family)
- >60 types
- Cannot be cultured
- Humans are the only known reservoir

Multiple hyperkeratotic papules on the dorsal of the thumb

- Warts may affect **any cutaneous or mucosal surface**
- **Different appearance:**

✓ Involved sites
✓ Type of infecting HPV
✓ The immunologic makeup of the host

Acquired by contact with an infected person. In the presence of:

1. **Breaks in the skin**
2. **Host's susceptibility** (not everyone exposed to the virus will get the disease only some people are susceptible to develop warts)

Types: (see next page)

1. Common warts
2. Flat warts
3. Plantar warts
4. Genital warts
5. Others

Investigations:

✓ Diagnosis is usually clinical
- Skin biopsy
- PCR detection and typing of HPVs

Common warts:

Note: warts papules are not confluent (remain separate and do not merge although they are adjacent).

Flat warts (plane warts):

They are skin color (or pigmented), small, flat growths.

seen more in children and don't require destructive measures. can be treated with salicylic acid or Tretinoin (retinoid).

Plantar warts:

- ✓ planter warts are **deeper** due the pressure of body weight, which makes them more difficult to treat (require more extensive destructive measures)
- ✓ only chronic planter warts cause pain (acute are painless)

Genital warts:

-The most common STD

-Condylomata accuminata

-Cauliflower like

Penile, vulvar skin, perianal area

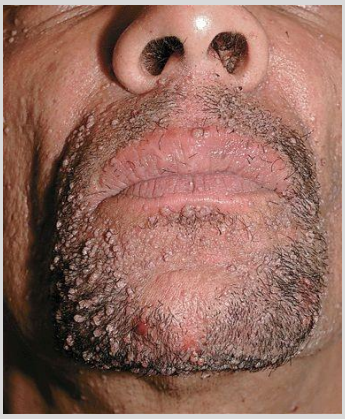
-Oncogenic type (16, 18, 31, 33) increase the risk of cervical cancer in females.

-genital warts have less keratinization (less scaly) because the area is moist; hence they have a more fleshy color.

Other:

Filiform warts:

Are pedunculated growths, commonly seen on the face and neck. can be misdiagnosed.



Treatment options include:

- Observation
- Topical salicylic acid preparations
- Destructive measures:
  1. Cryotherapy
  2. Electrosurgery or electrocautery
  3. Curettage
- Others (Imiquimod is classically used for genital warts but can be used for common and flat warts alone or in combination with cryotherapy).
- Best method of genital warts treatment is combination treatment (Cryotherapy + Imiquimod)

Note:

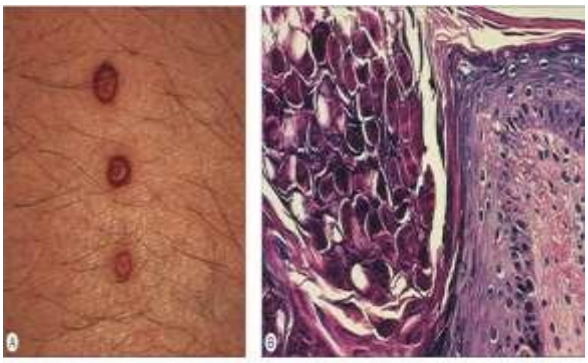
warts need to be taken more seriously when they show up for the first time in an adult with no history of exposure as they can be a sign of immunodeficiency.

Molluscum Contagiosum:

- Common viral skin disease caused by a DNA **poxvirus**
- Children > adults
- Presents as asymptomatic smooth surface, skin colored, translucent papules several millimeters in diameter with a characteristic central umbilication **(pearly shiny papules)**
- the secondary lesion seen on is molluscum dermatitis and is a result of immune reaction to the infection.
- Common sites: **face, trunk, extremities**
- Acquisition & resolution significantly affected by immunologic factors
- **Children with Molluscum:**
  ✓ Spontaneous involution in 6-12 months

Numerous papules on exposed sites ✓

- Hundreds or thousands of lesions on immunocompromised patients.
- **Diagnosis usually clinical** but when lesion is not clear we take biopsy for H/E



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Histological finding:

distinct virus inside macrophages (**Hunderson-patterson bodies**)

- ***Treatment options:***
  1. Observation
  2. Destructive

Herpes simplex:

Herpes Simplex Virus (HSV) infection is a common acute, self-limited usually recurrent eruption that characterized by small grouped vesicles on a red base.

✓ medical, cosmetic procedures (e.g. laser hair removal) or sun exposure can trigger recurrent herpes episode.

Etiology:

- Herpesvirus dominans(Ds-DNA)
- **Primary infection followed by latency**.
- Acquired by direct contact
- Types:

1. HSV-1 oral labial infection
2. HSV-2 genital infection

A- Primary oral HSV

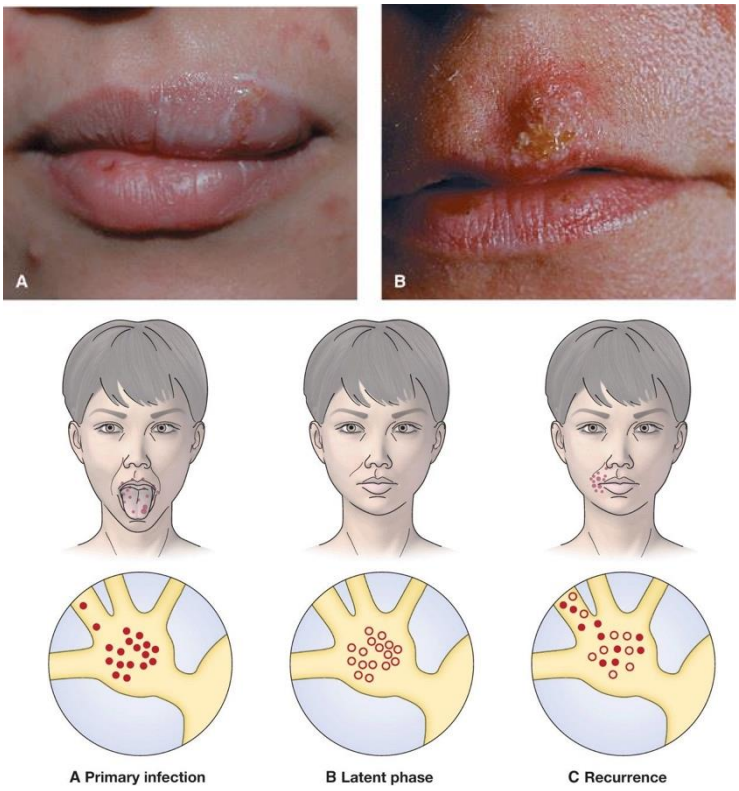
- HSV-1 > HSV-2
- Usually **occurs in children**
- May be subclinical
- Acute **gingivostomatitis** (commonest)
- Typically presents as crops of clear to yellow vesicles on erythematous background crust
- Sites: face, lips, palate, tongue
- **2 weeks course**

B- Primary Herpes Genitalis

- HSV-2 > HSV-1
- Usually **acquired after sexual contact**
- Presents with multiple erosions on the external genitalia
- **papule → vesicle → erosions**
- Healing in 2-3 weeks
- Associated: severe pain, dysuria, inguinal LAP
- >50%: fever, headache, malaise

C- Recurrent HSV infection

- HSV-2 > HSV-1
- Prodrome of tingling and stinging
- Reduced local symptoms, viral shedding and healing time than primary disease
- Frequency decreased with time



Complications:

1. Superimposed bacterial infections
2. Eczema herpeticum
3. Herpes encephalitis
4. Erythema multiforme

Eczema herpeticum:

Infection with HSV in patients with previous skin diseases (e.g. atopic dermatitis) with or without systemic symptoms (fever, malaise)

Erythema multiforme could develop into Steven Johnson syndrome

Diagnostic tools:

1. The Tzanck preparation
2. Immunofluorescent testing
3. Tissue cultures

Treatment:

- Pt education
- Severe oral/genital HSV
  - ✓ **Oral anti-viral** (acyclovir)
  - ✓ No effect on recurrences
- I**.V. antiviral treatment**
  - ✓ Immunosuppressed pts
  - ✓ Eczema herpeticum
  - ✓ Severe primary genital HSV
- Oral continuous suppressive oral anti-viral treatment for pts with frequent recurrences
- R/O other STDs (genital HSV)

Herpes Zoster:

- An acute self-limited disease characterized by painful small grouped vesicles on an erythematous base and usually localized to one or two dermatomes
- Incidence increasing with advancing age
- Results from **reactivation of latent Varicella-Zoster Virus** (VZV)

Clinical stages:

1. Prodrome (1-4 days) (burning or tingling sensation)
2. Vesicular stage
3. Crusted stage
4. Post herpetic neuralgia (pain after the lesions resolve)

Diagnostic tools:

1. The Tzanck preparation
2. Immunofluorescent testing
3. Tissue cultures

Treatment:

1. Symptomatic treatment
2. Oral anti-viral agent (Acyclovir) **(High dose within 48-72 hrs)**
3. ? Oral corticosteroids (May decrease acute pain, may reduce risk of PHN)

In infants and neonates, it can result secondary to mother's varicella infection (the virus is transmitted in-utero).

Varicella (chicken pox):

- Varicella is a **highly contagious disease of childhood** & occasionally adulthood caused by a primary infection with the VZV (varicella zoster virus)
- Transmitted by close contact and droplet infection
- **90% of cases occur by the age of 15 yr**

clinically: Successive crops of pruritic lesions on the trunk, face and scalp "few drops on a rose petal"

macule→ papule→ vesicle→ crust [all stages of development in the same anatomical area at the same time]

Infectivity:

1-2 days prior to the rash up to 5-7 days after the rash

Diagnostic tools:

1. The Tzanks preparation
2. Tissue cultures

Treatment:

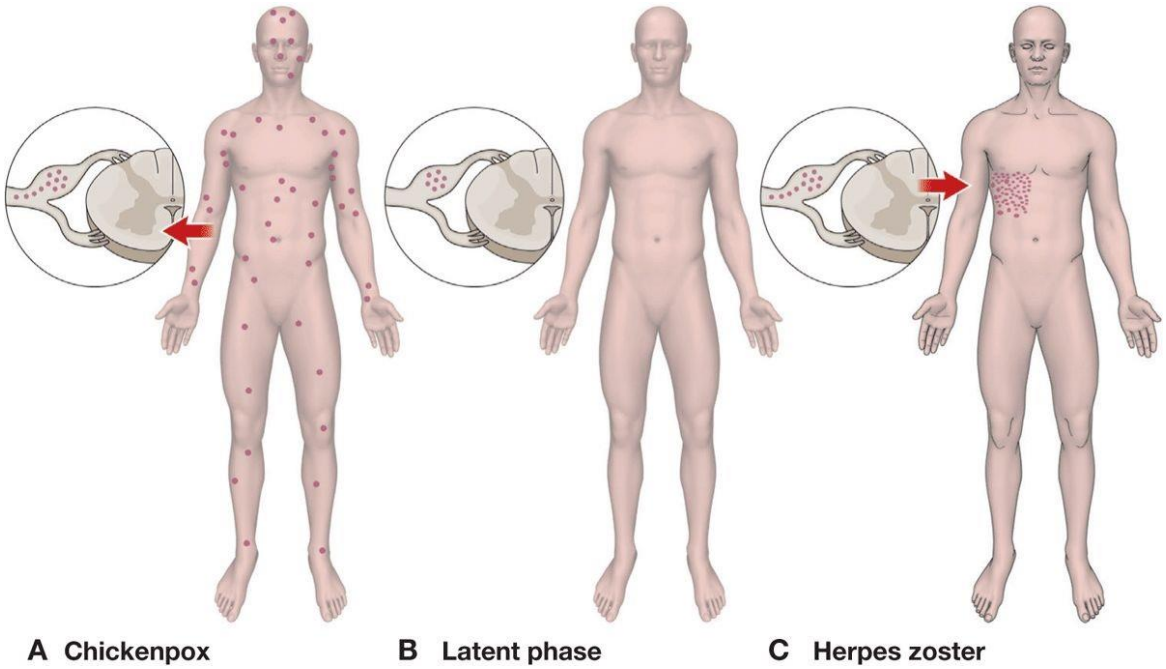
- **Symptomatic**
- Children
  - ✓ Benign disease
  - ✓ Avoid aspirin (risk of Reyes syndrome)

Varicella in adults:

- Prodrome, extensive rash
- More constitutional symptoms
- Predisposition to more severe complications.

- **Early high-dose systemic anti-viral** (Controversial in uncomplicated childhood varicella)
  - ✓ Immunocompromised
  - ✓ Varicella pneumonia

- VZIG (in exposed adults with no history of disease)
- Live attenuated vaccine: available



MCQs:

1- Impetigo is more common in:

A. Elderly

B. Children

C. Adults

2- which of the following has Oncogenic potential:

A. (HPV 1,3)

B. (HPV 16,18)

C. (HPV 31,33)

D. B&C

3- The initial symptom suggestion of herpes zoster is:

A. Dermatomal ulceration

B. Fever

C. Headache

D. Pain in dermatological distribution

4- 8 year old boy is complaining of pruritic lesions all over his body. He reports that his cousin has the same condition. The physical examination shows clusters of macules, papules, and vesicles with crust all over his trunk, upper and lower extremities. What is the diagnosis:

A. Herpes zoster

B. Impetigo

C. furuncle.

D. varicella zoster.

1-B 2-D 3-D 4-D

Done By:

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