Dermatology

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## Superficial Fungal Infections

### 1- Dermatophytoses

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<th>Epidermomycosis</th>
<th>Trichomycosis</th>
<th>Onychomycosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dermatophytosis of epidermis</td>
<td>Dermatophytosis of hair &amp; hair follicle</td>
<td>Dermatophytosis of nail apparatus</td>
</tr>
</tbody>
</table>

**Tinea Corporis**
- **Caused by:** *Trichophyton rubrum* (is most frequent)
- **Risk factors:** Typically seen in hot, humid climates
- **Lesion:**
  - Large, scaling, well-demarcated plaques (Single or scattered multiple lesions).
  - Peripheral enlargement & central clearing produce a cricinate pattern with concentric rings lesions.
- **Diagnosis:** clinically +
a skin scraping & KOH examination → microscope:
Dermatophytes are recognized as septated, tubelike structures (hyphae)
- **Treatment:** Topical antifungals is preferred treatment

**Tinea Capitis**
- **Caused by:** *T. tonsurans* (90%)
- **Lesions:**
  - 1- Non-Inflammatory lesions:
    - A) Scaly:
      - One, round, fine scally sharply marginated "grey patch" with partial alopecia (off-broken hairs)
    - B) Black dots:
      - Broken-off hairs at scalp surface give appearance of “dots” in dark-haired patients within alopecic areas.
  - 2- Inflammatory lesions:
    - C) Kerion:
      - Extremely tender boggy, purulent, inflamed nodules & plaques
- **Treatment:** Systemic antifungals with Griseofulvin

**Tinea Unguium**
- **Caused by:** *Trichophyton rubrum*
- **Lesions:**
  - 1- Distal subungual onychomycosis:
    - Presents as onycholysis, subungual debris and discoloration beginning at the hyponychium that spreads proximally.
  - 2- Proximal subungual onychomycosis:
    - Begins under underneath the proximal nail fold.
- **Treatment:** Systemic antifungals

### 2- Candidiasis

**Etiology:** *Candida albicans.* An oval yeast

**Ecology**
- Candidiasis is usually an endogenous infection.
- *C. albicans* & other species are a commensal organism in the mouth & GIT → it causes opportunistic infection.
- **KOH examination:** Budding yeast forms AND sausage-like pseudohyphal forms.

**Predisposing factors**
- Immuno-compromise
- Diabetes mellitus
- Obesity
- Hyperhidrosis, heat, maceration
- Systemic & topical glucocorticoids
- Chronic debilitation
- Pregnancy
- Moist opposing skin folds.

<table>
<thead>
<tr>
<th>Cutaneous candidiasis</th>
<th>Chronic candidal paronychia</th>
<th>Mucosal candidiasis</th>
</tr>
</thead>
</table>
| 1- **Intertrigo** (Erythema. Pruritus, tenderness, pain.)
  - Increased temp. & moisture in cutaneous folds make them susceptible.
| - Usually seen in wet workers & house wives.  
  - Nail fold: boggy, swollen, erythematous, tender on pressure.  
  - Nail plate may become irregular & discolored. | 1- **Oro Candidiasis (thrush):**
  - Adherent white patches on the tongue and inner surface of cheeks, if scraped off a raw bleeding area will be revealed.  
  - Seen with prolonged use of antibiotics or corticosteroids.  
  - It's maybe painful & interfere with eating.
| 2- **Interdigital** Most common in obese elderly.
  - **Distribution :**
    - Hands: usually between 3rd & 4th fingers.
    - Feet: maceration in webspace
| b- **Vulvo-vaginitis:**
  - Erythematous inflamed mucous membrane with white adherent plaque associated with itching & white creamy thick discharge.
| 3- **Diaper Dermatitis**
  - Erythema, edema with papular and pustular lesions; erosions, oozing, collarette-like scaling at the margins of lesions |
| a- **Monilial balanitis:**
  - Tiny white pustule or papules with soreness and irritation.
| Treatment:
  - **Topical antifungal agents:**
    - Nystatin cream: Effective for *Candida* only
    - Imidazole creams
      - Effective for candidiasis, dermatophytosis, pityriasis versicolor.
| **Oral antifungal agents:**
  - Azoles (fluconazole & itraconazole):
    - Treat cutaneous infection.
  - Nystatin: Eradicates bowel colonization.
| **Treatment:** Systemic antifungals

**Topical antifungal agents:**
- Nystatin cream: Effective for *Candida* only
- Imidazole creams
  - Effective for candidiasis, dermatophytosis, pityriasis versicolor.

**Oral antifungal agents:**
- Azoles (fluconazole & itraconazole):
  - Treat cutaneous infection.
- Nystatin: Eradicates bowel colonization.
3- Pityriasis Versicolor

**Etiology:** *Malassezia globosa* → Lipophilic yeast that normally resides in the keratin of skin & hair follicles (An opportunistic organism)

**Predisposing Factors:**
- Warm season or climates; tropical climate.
- Hyperhidrosis; aerobic exercise.
- Glucocorticoid treatment. • Immunodeficiency.

**Skin lesions:**
- **Macules** which may be confluent together forming patches, sharply marginated, varying in size.
- **Fine scaling** is best seen by gently scraping of the lesions with the edge of glass slide.
- **Color:** 1- Hypopigmented 2- Hyperpigmented

**Investigations:**
1) **Direct Microscopic:**
- Examination of Scales Prepared with KOH: Filamentous hyphae & globose yeast forms (termed *spaghetti & meatballs*), are seen.
2) **Wood Lamp:**
- Blue-green fluorescence of scales

**Treatment (Topical):**
- Selenium sulfide “lotion or shampoo”
- **Azole creams** (ketoconazole) Apply daily or twice daily for 2 weeks
- **Terbinafine:** 1% solution Apply twice daily for 7 days

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### Pityriasis rosea

- **Acute exanthematous eruption** with a **distinctive morphology** and often with **characteristic self-limited course**.
- **Caused by:** reactivation of HHV-7 or HHV-6, two closely related β-herpesviruses
- **Morphology:**
  1. Initial lesion: **“Herald” patch** develops usually on the trunk (Oval, slightly raised plaque or patch 2–5 cm, salmon-red color)
  2. Followed by: 1 or 2 weeks later a **generalized eruption** develops in a typical distribution pattern (“Christmas tree” pattern)
  3. **Spontaneous remission** in 6–12 weeks or less. (Recurrences are uncommon)

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### Scarlet Fever

- **Scarlet fever** is caused by strains of *Group A streptococcus* that produce **erythrogenic exotoxins**.
- **Mode of transmission & age of distribution:** as streptococcal pharyngitis:
  - Illness may follow a streptococcal pharyngitis, wound infections, burns, or streptococcal skin infection.
  - **Incubation period of 1 to 7 days**
  - Followed by the illness **acutely** with initial symptoms include: *fever, chills, toxicity + abdominal pain & pharyngitis*
  - **Within 12 to 48 hours,** rash initially appears on the neck, axillae, and groin.
  - Subsequently **generalizes within 24 hours**.
  - **Rash:** Punctate or finely Papular texture hence, the **“sandpaper-like” description**.
  - Rash is blanchable on pressure.
  - Rash fades by day 5–6 but residual petechial lesions are seen in cubital fossa (Pastia sign).
  - **Pharynx** is typically erythematous, swollen and possibly covered with **gray white exudates**, **Strawberry tongue** is also a feature
  - **Circumoral pallor:** area around mouth appears pale in comparison with extremely red cheeks
  - Towards the end of the first week, **desquamation (from face down to trunk finally to hands & feet)**

- **Treatment** is **Penicillin V (drug of choice)**
  - Erythromycin, Clindamycin, 1st generation Cephalosporins are good alternatives
<table>
<thead>
<tr>
<th>Other Childhood Exanthematous Rashes</th>
<th>Measels (Rubella)</th>
<th>Rubella (German measles)</th>
<th>Exanthema subitum (Roseola/HHV-6)</th>
<th>Erythema infectiosum (parvovirus B19)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cause:</strong></td>
<td>Rubella virus (RNA Paramyxovirus)</td>
<td>German measles</td>
<td>HHV-6</td>
<td>Parvovirus B19</td>
</tr>
<tr>
<td><strong>Prodrome:</strong></td>
<td>After 10 days: 1- High grade fever/anorexia with: 2- Non-productive cough (Bronchitis) 3- Coryza (Rhinitis) 4- Non-purulent Conjunctivitis 5- Koplik’s spots (pathognomonic)</td>
<td>After 14-21 days: 1- Low grade fever with : 2- Conjunctivitis 2- Coryza 3- Cervical Lymphadenopathy 4- Forschheimer spots (rose spots appear on soft palate before rash)</td>
<td>Abrupt high grade fever for 3-4 days</td>
<td>Very mild, or no fever</td>
</tr>
<tr>
<td><strong>Pathognomonic:</strong></td>
<td>Koplik’s spots: Blush-white lesions appear on erythematous buccal mucous membranes, opposite to 1st &amp; 2nd upper molars -- sometimes on - inner conjunctivae &amp; - vaginal mucosa</td>
<td>Tender and enlarged: 1- Occipital LNs 2- Posterior-auricular LNs Posterior cervical &amp; posterior auricular lymphadenopathies are common</td>
<td></td>
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</tr>
<tr>
<td><strong>Exanthem:</strong></td>
<td>1- Blanching reddish-brown maculopapular rash 2- Cephalocaudal &amp; centrifugal spread: Starts on the face behind ears, 24 hours then to neck → trunk → arms → lower limbs</td>
<td>1- Cephalocaudal spread of blanching erythematous maculopapular rash (starts on face &amp; then spreads to trunk &amp; limbs) - Rash is lasting for less than 3 days</td>
<td>1- Fever subsides on 5th day then: 2- Centrifugal spread of maculopapular rash (starts on trunk and then spread to extremities sparing the face)</td>
<td>Sudden appearance of “Slapped Cheeks” rash: - Maculopapular - Pruritic - Worsen with fever &amp; sun Exposure</td>
</tr>
<tr>
<td><strong>Diagnosis:</strong></td>
<td>Mainly clinical 1- PCR 2- Serology for anti-measles IgM &amp; IgG → 4 fold increase in titres 3- Lab findings include: leukopenia &amp; lymphopenia</td>
<td>Mainly clinical 1- PCR 2- Serology for: anti-rubella IgM &amp; IgG</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Complications:</strong></td>
<td>1- Otitis Media 2- Pneumonia 3- Neurologic: - Encephalitis - Acute disseminated encephalomyelitis (within weeks) - Subacute sclerosing panencephalitis (within years) 3- Gastroenteritis</td>
<td>1- Encephalitis 2- Thrombocytopenia 3- Congenital rubella syndrome: (Risk is high in 1st trimester) - Sensorineural hearing loss - Intellectual disability - Cardiac anomalies (PDA) - Cataract - Glucoma</td>
<td>Most common cause of febrile fits from rapid fever onset</td>
<td></td>
</tr>
<tr>
<td><strong>Prevention:</strong></td>
<td>Live attenuated measles vaccine</td>
<td>Live attenuated rubella vaccine</td>
<td></td>
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</tr>
<tr>
<td><strong>Treatment:</strong></td>
<td>1- Supportive care 2- Vitamin A for hospitalized children - Vitamin A has been shown to: a- reduce the morbidity &amp; mortality rates of patients with measles through immune enhancement. b- It also helps the gastrointestinal and respiratory epithelium to regenerate.</td>
<td>1- Supportive care: Acetaminphen</td>
<td></td>
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<td></td>
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<td>Patients can be infectious from 1 week prior to the onset of rash to 15 days after</td>
</tr>
</tbody>
</table>
Varicella: *Herpes zoster (shingles)* is the reactivation of VZV and occurs in dermatomal distribution

- Highly contagious, self-limited viral infection *caused by:* *Varicella-zoster virus (VZV)*, group of herpesviruses.
- Often, there is a history of exposure to infected individual, 90% of patient < 10 years old child.

**Prodrome:** Fever/Anorexia/Headache/malaise/ abdominal pain before the onset of the rash.

**Rash:**
- Erythematous macules, papules, vesicles, scabbed lesions are **present at the same time** → Within days, vesicles become turbid and then crusted.
- There is **centripetal distribution:** First on face and spread to trunk and extremities. *(sparring palms & soles)*

**Diagnosis:** is mainly clinical: PCR test of swab from the vesicles.

**Complications:**
- Skin lesions may be superinfected by bacteria (Streptococcus pyogenes or Staphylococcus aureus).
- Pneumonia in immunocompromised or pregnant patients.
- Encephalitis.
- Reye syndrome *(associated with aspirin use)*

**Treatment:**
1. For most immunocompetent children: *Symptomatic for fever and pruritus*
2. For VZV pneumonia and for immunocompromised individuals: *Acyclovir*

**Post-exposure prophylaxis:**
- Patients age **more than 1 year** who are:
  1. **Nonimmune**
  2. **Asymptomatic**
  3. **Immunocompetent**

  → should receive the **varicella vaccine** for post-exposure prophylaxis in < 5 days.

  - Vaccine is **70%-100%** effective in preventing infection if given **within 3-5d. of exposure**

- Patients who are:
  1. **Nonimmune**
  2. **Immunocompromised**

  → should receive **passive with varicella zoster immunoglobulin (VZIG)** within **10 days of exposure** as they are **at risk for life-threatening complications** → VZIG may **prevent infection BUT it can reduce severity** → So, require close monitoring for development of varicella infection as VZIG can **prolong the incubation period beyond 1 month.**

- Because the varicella vaccine is a live attenuated virus preparation, it is **contra indicated** in: 1- Pregnant women 2- Immunocompromised hosts.

**Rosacea:**
- **Age:** most commonly occurs in 30- to 60-year-old patients with fair skin, light hair and light eye color.
- **Pathogenesis** is not known, although hair follicle mites have been thought to play a role.
- It’s **chronic skin infection** that is characterized by: a **rosy hue with telangiectasia over cheeks, nose, chin**

  Sometimes, papules and **pustules** may be present

- **Precipitations:** Flushing of these areas is typically precipitated by:

- **Course:** The episodes are usually **intermittent**, but can progressively lead to permanently flushed skin.
- **Treatment:** 1) **Medical:** is aimed at the inflammatory papules, pustules, and erythema.
  2. **Laser surgery:** for telangiectasias

**Henoch-Schonlein purpura**
- A common vasculitic condition of childhood.
- It is commonly seen *after an upper respiratory tract infection* / and more common in males.
- The classic findings are:
  1. **Palpable purpura** in lower extremities & buttocks.
  2. **Renal findings:** hematuria & proteinuria.
  3. **Arthralgias:** most commonly affect the knees & ankles.
  4. When patients present with **abdominal pain**, the two common pathologies which should be ruled out emergently are: 1- GI bleeding 2- Intussusception

  → These symptoms are **always transient**, and there is **no** permanent damage to the joints

  **Treatment** includes: 1- administration of steroids
  2- monitoring of renal function.

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**Varicella post-exposure prophylaxis**

- **History of immunity** *(prior infection or vaccination)*
- **No** → Observation
- **Yes**
  - Immunocompromised → VZIG within 10 days of exposure
  - Immunocompetent → Varicella vaccine

**Henoch-Schonlein purpura**

<table>
<thead>
<tr>
<th>Pathogenesis</th>
<th>IgA-mediated leukocytoclastic vasculitis</th>
</tr>
</thead>
</table>
| Clinical manifestations | Palpable purpura  
Abdominal pain, intussusceptions  
Renal disease similar to IgA nephropathy |
| Laboratory findings | Normal platelet count & coagulation studies  
Normal to ↑ creatinine  
Hematuria +/- RBC casts +/- proteinuria |
| Treatment | Supportive (hydration & NSAIDs) for most patients  
Hospitalization & systemic glucocorticoids in patients with severe symptoms |

RBC = red blood cell; NSAIDs = nonsteroidal antiinflammatory drugs.
# Bacterial Infections

## 1- Superficial epidermal Infections

### Intertrigo

- **Clinical findings**: Erythema ± symptoms of pruritus, tenderness, or increased sensitivity

### Causes:
- **Bacteria**: Group A streptococcus, group B streptococcus, C. minutissimum (Erythrasma), P. aeruginosa

## 2- Soft tissue infections

### Cellulitis

- **Etiology**: Staphylococcal aureus & Group A beta-hemolytic Streptococci

### Clinical Features:
- **Systemic signs**:
  - High-grade fever with rigors & malaise, fatigue, confusion

### Erysipelas

- **Etiology**: Group A beta-hemolytic Streptococci (GAS)

### Clinical Features:
- **Local signs**:
  - Erythematous, warm, tender, oedematous, indurated
  - Less well-demarcated than erysipelas

### How can you differentiate erysipelas from cellulitis?

**Cellulitis is characterised by**:
- Lesions are primarily NOT raised.
- Ill-defined border

**Erysipelas**
- Raised
- Sharply-demarcated PLAQUE

- In severe cases, overlying epidermis may show: Bullae, Pustules or Necrosis.

### Treatment:
- **When systemic signs are present**: IV Nafcillin or Cefazolin
- **In areas with high prevalence of MRSA**: Vancomycin

## Necrotizing fasciitis

- **Causes**: 1. It usually occurs after trauma 2. It may also occur around foreign bodies (in surgical wounds). 3. It can be idiopathic (e.g., scrotal or penile necrotizing fasciitis).

### Organisms:
- Group A hemolytic Streptococci & Staphylococcus aureus

### Clinical features:
- **Signs of systemic toxicity** (e.g., fever, hypotension) may be present.
- There is a sudden onset of pain and swelling at the site of trauma or recent surgery, progresses to purplish discoloration & gangrenous changes.
- A gloved finger can easily be passed between the 2 layers, revealing yellowish green necrotic fascia, which helps in the diagnosis.

### Investigations:
- **CT scan** is useful in identifying the involved site, which reveals: 1. necrosis 2. asymmetrical fascial thickening 3. gas in the tissues.

### Treatment:
- **Thorough surgical debridement** of all the necrotic tissues is the most important therapy.
- **High-flow oxygen, fluid resuscitation, and broad-spectrum antibiotics** should be included in the management.
### Impetigo

**Definition:**

A superficial pyogenic skin infection originating in the epidermis (impetigo)

**Etiology:**

- Staphylococcal aureus (most commonly)
- Group A beta-hemolytic Streptococci

**Types:**

1. **Vesiculo-Pustular (Non-bullous) impetigo**
   - **Effects:**
     - Superficial infections: in minor superficial breaks in the skin
     - Secondary infections of preexisting dermatoses (impetiginization or 2nd infection)

2. **Bullous impetigo:** neonates, especially; children <5 years old.

**Etiology:**

- Group A beta-hemolytic Streptococci

**Clinical features:**

- Hard bullae surrounded by erythematous skin
- **Bacteriology:**
  - Staphylococcal Scalded Skin Syndrome (SSSS): epidermolytic toxin A & B
  - Bullous impetigo: epidermolytic toxin A (etA) gene producing S. aureus, which also causes Staphylococcal Scalded Skin Syndrome (SSSS).

**Complications:** may post-streptococcal glomerulonephritis.

**Predisposing factors:**

- Warm and humid climate
- Poverty
- Crowding
- Poor personal hygiene
- Nasal carriage of GABS or S. aureus.
- **Epidermolytic toxin A** can cause recurrent impetigo.

**Location:**

- Initially: Painful, solitary, erythematous, deep seated, follicular NODULE.
- Nodule becomes fluctuant, with ABSCESS formation
- A variable zone of cellulitis may surround the furuncle.

**Treatment:**

- **Topical mupirocin** is the treatment of choice
- Alternatives: 1. Oral Erythromycin
  - Oral Cephalexin / Dicloxacillin / Ampicillin in severe cases.

**Folliculitis**

**Definition:**

Pyogenic infection of hair follicle with pus in the ostium of follicle.

**Etiology:**

- **Staphylococcal aureus** (MSSA, MRSA).

**Classification:**

- **Superficial** → Acute: Bockhart’s impetigo
- **Deep** → Acute: Furuncle/Carbuncle

**Pathogenesis:** They represent a continuum of severity of S. aureus infection. Portal of entry: hair follicle, break in the integrity of skin.

**Furuncle**

- **Complications:**
  - May cause post-streptococcal glomerulonephritis (etA) gene producing S. aureus, which also causes Staphylococcal Scalded Skin Syndrome (SSSS).

**Carbuncle**

- Evolution is similar to that of furuncle BUT:
  - Composed of several to multiple, adjacent, coalescing furuncles

- **Sycosis barbae**
  - Discrete follicular pustules, each pierced by a hair, in the beard.
  - Crustation follows, ending by scarring.
  - New lesions develop as the old ones heal.

**Pseudo folliculitis**

- Erythematous papulo-pustules, with skin-buried hair.
- It affects mainly the beard of blacks.
- It results from ingrowing hair, which predisposes to 2nd infection, caused mainly by *Staph. aureus.*

**Portal of entry**

- Primary infections in minor superficial breaks in the skin
- Secondary infections of preexisting dermatoses (impetiginization or 2nd infection)

**Age of Onset:**

- Primary infections: more common in children
- Secondary infections, at any age.

- **Bullous impetigo:** neonates, especially; children <5 years old.

**Clinical features:**

- Hard bullae surrounded by erythematous skin
- **Bacteriology:**
  - S. aureus (most commonly)
  - Group A beta-hemolytic Streptococci

**Complications:**

- May cause post-streptococcal glomerulonephritis.

**Predisposing factors:**

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- Poverty
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- Poor personal hygiene
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- **Topical mupirocin** is the treatment of choice
- Alternatives: 1. Oral Erythromycin
  - Oral Cephalexin / Dicloxacillin / Ampicillin in severe cases.
Staphylococcal Scalded Skin Syndrome (SSSS)

- **Age:**
  - SSSS is primarily a disease of children but adults with renal disease or immunocompromise may also be affected.
  - It is usually seen in children less than 6 years of age.

- **Caused by:** exfoliative toxin-producing strains of *Staphylococcal aureus* → The toxins target desmoglein 1, which is responsible for keratinocyte adhesion in the superficial epidermis.

- **Prodrome:** of 1- fever 2- irritability 3- skin tenderness

- **Eruption:** Erythema starts on the face, and generalizes within the ensuing 24-48 hours → Superficial flaccid blisters soon develop, with flexural accentuation and perioral crusting.

- **Nikolsky sign is positive** (gentle lateral pressure on the skin surface adjacent to a blister causes slipping & detachment of a superficial layer of skin).

- **Eruption:** The blisters of SSSS are fragile and when unroofed reveal a moist erythematous base.

- **Cultures from intact bullae are usually sterile because this is a toxin-mediated process.**

- **Treatment:**
  1. Eliminate any inciting focus of infection with appropriate anti-staphylococcal antibiotics
  2. Provide supportive wound care of all denuded areas.

- **Felon:**
  - Tailors can develop felon due to needle injuries.
  - Clinical features: Felon is a bacterial infection of the distal volar space, characterized by a tense abscess and intense throbbing pain.
  - Treatment: Incision & drainage with appropriate antibiotic (e.g., cephalosporins) is the treatment of choice.

- **Systemic Inflammatory Response Syndrome (SIRS):**
  - **Pathophysiology:** There is usually an original insult (e.g., infection and injury) that leads to inflammation & a dysregulated host response, with a massive and uncontrolled release of proinflammatory substances causing extensive tissue damage. → This response to an infection is referred to as sepsis. Some noninfectious causes are known as systemic inflammatory response syndrome (SIRS), which:
    - Temperature >38.5°C (101.3°F) or <36°C (96.8°F)
    - HR >100/min
    - Respiratory rate >20/min
    - WBC >12,000 cells/mm³, <4,000 cells/mm³, or >10% bands
  - SIRS is defined as having at least 2 of the following criteria →
  - It can occur in: pancreatitis, autoimmune disease, vasculitis, burns.

- **Sepsis (i.e., SIRS with a known infection):** Criteria that indicate sepsis in the patients include:
  1. Worsening hyperglycemia (due to worsening insulin resistance),
  2. Leukocytosis,
  3. Thrombocytopenia
  4. Mild hypothermia (temperature <36C),
  5. Tachypnea, and Tachycardia.

- **Sepsis is considered severe** when there is associated end-organ dysfunction, such as:
  1. Oliguria
  2. Hypotension (i.e., systolic <90 mm Hg)
  3. Thrombocytopenia (i.e., platelet count <80,000/mm³)
  4. Metabolic acidosis
  5. Hypoxemia.

- **Complications of burns in 1st week:**
  1. Severe burns → manifest some evidence of SIRS → sepsis & Septic shock from wound infections (S. aureus or P. aeruginosa).
  2. Hypermetabolic response:
    1. Hyperglycemia (due to insulin resistance)
    2. Muscle wasting
    3. Protein loss
    4. Hyperthermia
    5. Increased energy expenditure.

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## Viral Infections

### 1- Human Papilloma Virus (HPV) infections "Warts"

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<tr>
<th>Clinical type</th>
<th>Site</th>
<th>Presentation</th>
<th>Serotype</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Common</strong> (verruca vulgaris)</td>
<td>Hands</td>
<td>- Hyperkeratotic, scaly, rough, skin-coloured papules.</td>
<td>2,4,29</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Characteristic “red or brown dots” are pathognomonic, representing thrombosed capillary loops.</td>
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<tr>
<td></td>
<td></td>
<td>- painless – Usually multiple.</td>
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</tr>
<tr>
<td><strong>Plantar</strong> (v. plantaris)</td>
<td>Soles</td>
<td>Early small, sharply marginated papule → plaque with rough hyperkeratotic surface, studded with brown-black dots (thrombosed capillaries).</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Tender – single or multiple.</td>
<td></td>
</tr>
<tr>
<td><strong>Flat</strong> (v. plana)</td>
<td>Face &amp; hands</td>
<td>Small (2-5 mm), sharply defined, flat-topped, skin-colored or hyperpigmented papules.</td>
<td>3,10</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- They are multiple.</td>
<td></td>
</tr>
<tr>
<td><strong>Anogenital</strong> (Condyloma Acuminatum)</td>
<td>Anogenital region</td>
<td>Moist, exophytic (cauliflower-like), papules/nodules, variably sized; One of Sexually Transmittes Diseases.</td>
<td>6,11</td>
</tr>
</tbody>
</table>

### Treatment

<table>
<thead>
<tr>
<th>Chemical</th>
<th>Common, Planter Flat External genital</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Salicylic acid (Keratolytic)</td>
<td>For small lesions 10–20% / For large lesions 40% for 1 week</td>
</tr>
<tr>
<td></td>
<td>5-fluouracil / Tretinoin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Imiquimod cream / Podophyllotoxin</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Surgery</th>
<th>All types</th>
<th>Cryosurgery: using cryospray, freezing the wart and 1–2 mm of surrounding normal tissue for approximately 30 s (Don't use in face as it cases scarring)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Resistant</td>
<td>Electrocautery: More effective than cryosurgery, but associated with scarring</td>
</tr>
<tr>
<td></td>
<td>Resistant</td>
<td>Surgical Excision</td>
</tr>
</tbody>
</table>

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2- Molluscum Contagiosum

- **Etiology:** Molluscum contagiosum virus (MCV), is a pox virus.
- **Description:**
  - It’s characterized by multiple, *dome-shaped lesions with central umbilication.*
  - Lesions typically have a pink, translucent quality and may be *arranged in a linear fashion* due to spread of the virus to adjacent areas.
  - It can be **generalized** in immunodeficient conditions (such as AIDS, especially when CD4 count < 100/ul).
  - Conjunctivitis (as seen in this patient) may occur if the lid margins have been infected.
- **Duration of lesions:** Is it self limited? **YES**, it usually persists up to 6 months, then undergo spontaneous regression.

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3- Herpes Simplex Virus (HSV) infections

**Etiology:** HSV-1 and HSV-2.

**Incubation Period:**
- HSV-1: 3-14 days (average 3-5).
- HSV-2: 7-10 days (average 5-7).

**Primary Herpes**

- **Constitutional symptoms:** Fever, headache, malaise, myalgia.
- **Skin findings:**
  - Sudden onset of grouped vesicles on erythematous base which may evolve to pustules.
  - They slough to form erosions which may enlarge to ulcerations which may be crusted.
  - Healing: in 2–4 weeks, with resultant hypo or hyper-pigmentation, uncommonly with scarring.
- **Mucocutaneous findings:**
  - Oral mucosa usually involved only in primary HSV infection at any site in the oropharynx.
  - Conjunctival & corneal autoinoculation may occur.

**Recurrence Herpes**

- **Prodrome of tingling, itching sensation precedes visible skin changes by 24 h.**
- **Constitutional symptoms are usually absent.**

**Herpes whitlow**

- It is a common **viral infection of the hand** → caused by either type 1 or 2 **herpes simplex virus**, and is usually **self-limiting**.
- **Mode of transmission:** Direct inoculation of the virus through broken skin.
- **Most commonly seen in:** 1- Women with genital herpes or 2- Children with herpetic gingivostomatitis.
  - 2- **Health care workers** are also at **increased risk** of this infection, due to contact with infected serum or saliva.

- **Clinical presentation:**
  - Systemic symptoms such as fever and lymphadenopathy may occur.
  - Patients often present with throbbing pain in the distal pulp space, which is swollen, soft and possibly tender.
  - Lateral nail fold may also be affected.
  - Non-purulent vesicles on the volar aspects are **clinically diagnostic**.
- **Diagnosis** is confirmed by 1- positive history of exposure & 2- multinucleated giant cells in the Tzanck smear of the vesicles. This
- **Treatment:** it is a self-limiting illness → however: 1- Oral acyclovir & 2- **topical bacitracin** to prevent secondary infection may be used.
### 4- Herpes Zoster Virus (HZV)

<table>
<thead>
<tr>
<th>Primary (Chickenpox)</th>
<th>Recurrent (Shingles)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patients affected</strong></td>
<td>More common in children</td>
</tr>
<tr>
<td><strong>Timing of presentation</strong></td>
<td>Symptoms 21 wk after infection occurs; Symptoms of headache, malaise, myalgia, and fever precede development of lesions by 3 days</td>
</tr>
<tr>
<td><strong>Type of lesion</strong></td>
<td>Small, red macules that evolve into papules And then vesicles that eventually become crusted</td>
</tr>
<tr>
<td><strong>Distribution of lesions</strong></td>
<td>Wide distribution</td>
</tr>
<tr>
<td><strong>Course of disease</strong></td>
<td>Lesions may develop up to 1 wk and resolve a few days after appearing; Lesions exist for a week and may be painful; infective until lesions crust over</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td></td>
</tr>
<tr>
<td>1. Antipruritics aid symptoms; Analgesics, possible corticosteroids; 2. Acyclovir used in immunocompromised patients and trigeminal nerve distribution; 3. Vaccination has reduced disease incidence significantly</td>
<td>2. Acyclovir used in severe cases or immunocompromised patients</td>
</tr>
<tr>
<td><strong>Complications</strong></td>
<td>1. Post-infectious neuralgia; treatment with antiviral agents decreases duration of symptoms &amp; incidence of post herpetic neuralgia; 2. Trigeminal neuropathy</td>
</tr>
</tbody>
</table>

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Acne Vulgaris

- An inflammation of pilosebaceous units, very common
- Appears in certain body areas (face, neck, trunk, back)
- Most frequently in adolescents
  - Acne usually decreases in severity as adolescence ends.
  - Corticosteroid use and androgen production disorders are common causes of outbreaks in adulthood
- Manifests as:
  1. Comedones:
     - Open comedones (blackheads)
     - Closed comedones (whiteheads)
  2. Papulopustules
  3. Nodules
- Results in pitted, depressed, or hypertrophic scars

<table>
<thead>
<tr>
<th>Comedonal acne</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Closed or open comedones on forehead, nose &amp; chin</td>
</tr>
<tr>
<td>• May progress to inflammatory pustules or nodules</td>
</tr>
<tr>
<td>• Treatment: Topical retinoids, salicylic, azelaic, or glycolic acid</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Inflammatory acne</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Inflamed papules (&lt;5 mm) &amp; pustules, erythema</td>
</tr>
<tr>
<td>• Treatment: Mild: Topical retinoids + benzoyl peroxide</td>
</tr>
<tr>
<td>• Moderate: Add topical antibiotics (eg, erythromycin)</td>
</tr>
</tbody>
</table>

- Coalescing nodules, cysts, abscesses, ulceration and irregular scars, producing pronounced disfigurement.

Aphthous ulcers

- They are described as shallow, fibrin-coated ulcerations with underlying mononuclear infiltrates.
- Aphthae (canker sores) are recurrent, self-limiting ulcerations of indeterminate (possibly autoimmune) etiology.
- Site:
  - These arise in the mucosa of the oral cavity, (NOT found in the vermilion zone of the lips or on the gingival)
  - NOT appear in surfaces covered by keratinized stratified squamous epithelium.

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### Eczema / Dermatitis

#### 1- Contact Dermatitis

<table>
<thead>
<tr>
<th>Irritant Contact Dermatitis</th>
<th>Allergic Contact Dermatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Most common form of occupational skin disease</td>
<td>Caused by an antigen (allergen) that elicits:</td>
</tr>
<tr>
<td>- It is caused by exposure to chemical or other physical agents that are capable of irritating the skin, acutely or chronically</td>
<td>Type IV (cell-mediated or delayed) hypersensitivity reaction.</td>
</tr>
<tr>
<td>Dependent on concentration of the offending agent</td>
<td>Host requires to be sensitized before a reaction will develop, so a patient must have had a prior exposure to the allergen before developing a reaction.</td>
</tr>
<tr>
<td>Occurs in everyone</td>
<td>Dependent on degree of sensitization:</td>
</tr>
<tr>
<td>- Confined to the area of exposure → therefore always sharply marginated &amp; never spreads</td>
<td>Minute amounts of the offending agents may elicit a reaction.</td>
</tr>
<tr>
<td><strong>Acute ICD</strong></td>
<td><strong>Chronic ICD</strong></td>
</tr>
<tr>
<td>- Skin findings :</td>
<td>- Reaction that tends to involve the surrounding skin (spreading phenomenon) and may even spread beyond affected sites</td>
</tr>
<tr>
<td>- Nonglistening surface → vesiculation (or blister formation)</td>
<td>~ Term: Allergic phytophotodermatitis (APD)</td>
</tr>
<tr>
<td>- Erosion → crusting → shedding of necrotic tissue</td>
<td></td>
</tr>
<tr>
<td>- Vesiculation:</td>
<td></td>
</tr>
<tr>
<td>- Erythema</td>
<td>-</td>
</tr>
</tbody>
</table>
An acute, subacute, or chronic relapsing skin disorder

**Age of onset:**
- usually begins in infancy from 2 months – 1 year (in 60% of cases) → **Infantile AD**
- begins by age of 5 years (in 30%)
- begins from 6 years – 20 years (in 10%)

**Risk factors:** History of atopy: 1- Asthma  2- Allergic rhinitis  3- Family history

**Pathophysiology:**
- Atopic dermatitis is the result of **decreased skin barrier function** due to improper synthesis of components of the epidermal cornified cell envelope → associated with 1- impaired filaggrin production, 2- reduced ceramide levels  3- increased transepidermal water loss
- This allows allergens ready access to the deeper levels of the epidermis where they may generate the immune response characteristic of atopic dermatitis (Type I (IgE-mediated) hypersensitivity reaction)

**Clinical features:**
- **Distribution:**
  1- Typically affects face, scalp, or flexural surfaces of the extremities, which is significantly from that seen in adults: mostly flexural surfaces)  2- The diaper region is typically spared.

**Treatment:**
1- Treatment is with improvement of skin barrier function through 1- the use of mild cleansers & 2- thick, bland emollients
2- In addition to moisturization, mild anti-inflammatory agents may be used.
3- Severe cases: treated with: 1- Oral corticosteroids   2- Anti-histamines  3- Seborrheic Dermatitis

**Chronic inflammatory papulosquamous disease occurring in regions where the sebaceous glands are most active.**

**Age:** can affect all age groups → found with increased frequency in patients with:
1- Parkinson disease  2- HIV → severe intractable SD should be a clue to the existence of HIV disease

**Pathogenesis:** Malassezia furfur is said to play a role in the pathogenesis, and response to topical ketoconazole & selenium sulfide is some indication.

**Clinical features:**
- **Distribution:**
  1- Head:
  - In infants: it often begins on scalp and called: "cradle cap": Erythema & yellow-orange scales & crusts on infants scalp, scalp (dandruff)
  2- Face: ("butterfly") areas
  - Areas most commonly affected: eyebrows, nasolabial folds, bases of the eyelashes (blepharitis) and paranasal skin
    **D.D:** Erythema of SD is often overlooked and thought to be the flushing of rosacea. **SD does not respond to ttt of rosacea**
  - Ears: retroauricular, meatus
  3- Trunk:
  - Yellowish-brown patches over the sternum common
    **D.D:** Simulating lesions of pityriasis rosea or pityriasis versicolor
  4- Body folds:
  - Axillae, groins, anogenital area, submammary areas, umbilicus, and diaper area

**Description:**
- Transparent to **yellow papules** and occasional **greasy looking scaling plaques** are characteristic

**Treatment:**
1- Moisturizers  2- Topical antifungals  3- Anti-dandruff shampoos  4- Topical steroids. **Severe cases may suggest underlying immunodeficiency.**
### Pemphigus Vulgaris

- **Phathogenesis:**
  - Loss of the normal cell-to-cell adhesion in the epidermis (acantholysis) occurs as a result of circulating Auto-Antibodies of the IgG class; which bind to desmogleins 3.

- **Clinical manifestations:** → 2 types: Localized / Generalized
  - Distribution:
    - PV usually starts in the oral mucosa (rapidly become erosive & painful preventing eating), and months may elapse before skin lesions occur.
    - Lesions may be localized for months, after which generalized bullae occur.

- **Description:**
  - It is characterized by flaccid bullae (blisters) that appear spontaneously → when rupture;
  - Early separation of the epidermis from the dermis → erosive lesion is characteristic.

- **Immunofluorescence microscopy reveals desmoglein 3 >> desmoglein 1**

- **Treatment:** Immunosuppressive agents
  - Commonly used drugs for treatment are steroids
  - Azathioprine may be used with prednisone & methotrexate

### Bullous Pemphigoid

- **Clinical manifestations:**
  - May presente bullae formation by months.
  - It is characterized by tense blisters, as opposed to the flaccid blisters in pemphigus.
  - Eruption may be localized or generalized.
  - Oral lesions are very rare.

- **Investigations:**
  - 1- Immunofluorescence microscopy reveals IgG & C3 deposits in the dermal epidermal junction.
  - 2- Antibasement membrane IgG autoantibodies in serum.

- **Treatment:** Immunosuppressive agents
  - In very mild cases and for local recurrences, topical glucocorticoid or topical tacrolimus
  - Systemic prednisone wither alone or combined with azathioprine
  - Low-dose MTX is effective & safe in elderly

### Dermatitis Herpetiformis

- **Clinical manifestations:**
  - A chronic, recurrent, intensely pruritic eruption occurring symmetrically on the extremities and trunk.

- **Associated with** gluten-sensitive enteropathy (GSE).

- **Investigations:**
  - 1- Immunofluorescence shows granular IgA deposits along dermal papillae.
  - 2- Circulating anti-endomysial antibodies can be detected in all patients.

- **Treatment:**
  1- Gluten-free diet &
  2- Dapsone are effective treatment options.
Rough Actinic keratoses develop in genetically predisposed individuals. Clinical features:

- Itching and confluent plaques result from the interaction of UVR and sensitivity to photosensitizing chemicals.
- It can be triggered by:
  - Sun exposure to certain drugs (via ingestion, IV, IM, or topical application) & to UVR
  - Ingestion, IV, IM, or topical application

Pathophysiology:

- Phototoxic drug reactions result from the production of reactive oxygen species by the interaction of drug and UVR.
- Prior sensitization is NOT required for phototoxic drug eruption.
- It is required for photosensitivity disease.

Treatment:

- Adverse reaction of the skin that results from simultaneous exposure to certain drugs (via ingestion, IV, IM, or topical application) & to UVR.
- Drugs cause phototoxicity: (FAST)
  - Fluoroquinolones / Aminoglycosides
  - Sulfonamides / Tetracyclines

Pathophysiology:

- Phototoxic drug reactions result from the production of reactive oxygen species by the interaction of drug and UVR.
- Prior sensitization is NOT required for phototoxic drug eruption.
- It is required for photosensitivity disease.

Pathology:

- The inflammatory response is a phototoxic reaction to photosensitizing chemicals in several plant families. Commonly due to exposure to:
  - 1. Sunlight
  - 2. UV radiation
  - 3. Erythema
    - Sunburn reaction: exposure to UVR from sunlight or artificial sources. It results in an inflammatory response and can be triggered by:
      - Sunlight
      - UV radiation
      - Photosensitizing chemicals

Classification of Skin Reactions to Sunlight

1- Phototoxicity

<table>
<thead>
<tr>
<th>Acute Sun Damage (Sunburn)</th>
<th>Phototoxic Drug Reaction</th>
<th>Plant-induced (phytophotodermatitis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sunburn is an acute, delayed, and transient inflammatory response of normal skin after exposure to UVR from sunlight or artificial sources.</td>
<td>Adverse reaction of the skin that results from simultaneous exposure to certain drugs (via ingestion, IV, IM, or topical application) &amp; to UVR.</td>
<td>Phytophotodermatitis (plant + light = dermatitis) is an inflammation of the skin caused by contact with certain plants during occupational exposure to sunlight.</td>
</tr>
<tr>
<td>Sunburn reaction:</td>
<td>Drugs cause phototoxicity: (FAST)</td>
<td></td>
</tr>
<tr>
<td>Erythema, if severe: edema, vesicles and bullae, frequently resolves with hyperpigmentation.</td>
<td>Fluoroquinolones / Aminoglycosides</td>
<td></td>
</tr>
<tr>
<td>Treatment:</td>
<td>Sulfonamides / Tetracyclines</td>
<td></td>
</tr>
<tr>
<td>1- Topical - Cool wet dressings, Topical glucocorticoids.</td>
<td>Pathophysiology:</td>
<td></td>
</tr>
<tr>
<td>Pathophysiology:</td>
<td>Photic drug reactions result from the production of reactive oxygen species by the interaction of drug and UVR.</td>
<td></td>
</tr>
<tr>
<td>Acute, delayed, and transient skin reaction in a photo distribution.</td>
<td>Prior sensitization is NOT required for phototoxic drug eruption.</td>
<td></td>
</tr>
<tr>
<td>Severe phototoxic reactions can occur within minutes of exposure and may exceed 10 mm.</td>
<td>In most patients the blistering drug has been applied topically.</td>
<td></td>
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<tr>
<td>Chronic allergic-like keratosis and extremely dry confluent plaques result.</td>
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<td>In most patients the blistering drug has been applied topically.</td>
<td></td>
</tr>
</tbody>
</table>

2- Metabolic & Nutritional

Porphyria cutanea tarda

It's a condition that arises from the deficiency of: hydropsynthetic pathway.

- It can be triggered by: the ingestion of certain substances:
  - (e.g., ethanol, estrogen) OCPs, that should be discontinued once suspected.
  - It is often associated with: Hepatitis C infection.

Clinical features:

- With repeated sun exposure and occurs after minor trauma
- 1. Painless blisters, increased skin fragility on dorsal surfaces of hands & feet
- 2. Facial hypertrichosis
- 3. Hyperpigmentation

Diagnosis:

- Is confirmed with: pinkish-red fluorescence in the urine when examined with a Wood lamp (urinary porphyrin).

Treatment:

- Phlebotomy or Hydroxychloroquine may provide relief

Pellagra

Because of Vitamin B3 (niacin) deficiency

Severe deficiency leads to: Pellagra: 3 D's of B3

- Diarrhea, Dementia, Dermatitis: 4

- Description:
  - Skin of sun-exposed limbs becomes indurated, lichenified, rough covered by dark scales & crusts

Distribution:

- Is striking:
  - 1. Butterfly region of the face
  - 2. C3/C4 dermatome circumferential "broad collar" rash [Casal necklace]
  - 3. Dorsa of hands and fingers ("gauntlet" of pellagra)
  - 4. Dorsa of feet up to malleoli with sparing of the heel

4- Chronic Photodamage

Actinic Keratosis

Actinic keratoses develop in genetically predisposed individuals 40-60 years of age under the influence of excessive chronic sun exposure.

Distribution:

- Most commonly affected areas are face, ears, scalp and the dorsa of the arms and hands, also legs, back & upper chest

Histology, affected areas show:

- 1. Acanthosis (thickening of the epidermis)
- 2. Parakeratosis (retention of nuclei in the stratum corneum),
- 3. Dyskeratosis (abnormal keratinization)
- 4. Hyperkeratosis (thickening of stratum corneum).

They are classically described as erythematous papules with a central scale due to hyperkeratosis. They may become prominent and turn into"cutaneous horns".

Course:

- Keratinocytes display various degrees of atypia. Actinic keratosis is regarded as either a premalignant condition or a carcinoma in situ, but fewer than 1% of AKs will evolve into frank squamous cell carcinoma.

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**Skin Cancer**

### Squamous Cell Carcinoma

- **The most likely diagnosis of:**
  1. Asymptomatic (i.e., lesion-free),
  2. In immunocompetent adult patient,
  3. With a significant history of sun exposure,
  4. With a [non-healing, isolated ulcer in the vermilion zone of the lower lip](#) is squamous cell carcinoma

- **Which is characterized by** invasive cords of POLYGONAL squamous cells with KERATIN pearls.

- **Risk factors:**
  1. Exposure to sunlight "UVB" (The single most important risk factor)
  2. Arsenic & aromatic hydrocarbons are other well-known factors associated with SCC
  3. Actinic Keratosis
  4. Significant history of alcohol & tobacco use:
    - [SCC of the mucosa of the mouth & neck is common in people with a significant history of alcohol & tobacco.](#)
    - Significant history of alcohol use is also a risk factor specific to palate SCC.

- **Distribution:**
  - > 90% occur in the face / rarely found on the lips.
  - Risk factors:
    - Significant history of alcohol & tobacco use:
    - [SCC of the mucosa of the mouth & neck is common in people with a significant history of alcohol & tobacco.](#)

- **Histologically:**
  - BCC histologically characterized by invasive clusters of spindle cells surrounded by palisaded basal cells.

- **Treatment:**
  - Most appropriate treatment:
    - Surgical excision using microscopically-controlled margins (Mohs technique).

### Basal cell Carcinoma

- **BCC** is the most common and the most malignant type of cancer in mankind.
- **Risk factors:**
  - It usually occurs in fair-skinned individuals with a history of prolonged sun exposure.
    - There are often telangiectasias overlying and at the periphery of the lesion.
    - Bleeding and ulceration are common features.

- **Distribution:**
  - Nodular: > 90% occur in the face (rarely found on the lips)
    - Develops in Eyelid (Most common location for eyelid BCCs is the lower eyelid margin) → thinning or loss of eyelashes in the region of the tumor is typical.
    - Spread from a periocular BCC into the orbit can occur and enucleation of the eye & required to treatment.

- **Histologically:**
  - BCC histologically characterized by invasive clusters of spindle cells surrounded by palisaded basal cells.

- **Treatment:**
  - Most appropriate treatment:
    - Surgical excision using microscopically-controlled margins (Mohs technique).

### Differential Diagnosis of Eyelid BCC: Chalazion

- This is a chronic granulomatous condition that develops when a meibomian gland becomes obstructed.
- Chalazion initially presents as a painful swelling that progresses to a nodular rubbery lesion.
- Persistent or recurrent chalazion may be due to meibomian gland carcinoma (sebaceous carcinoma).
- Basal cell carcinoma frequently presents as a solitary nodule on the lid margin and may initially be clinically difficult to distinguish from a chalazion. → Patient therefore requires histopathologic examination to rule out malignancy.

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Melanoma is now the most common malignancy in women aged 25 - 29 years old. It is second only to thyroid and breast cancer in the age groups flanking 25 - 29 years.

Risk factors:
- Fair skin types
- History of blistering sunburns
- Dysplastic nevus syndrome
- Atypical nevi & greater than 100 typical nevi.
- A recently changed mole is the strongest risk factor for malignancy.

Complaints: Patients often present complaining of:
- mole that has changed in size or color (darkening or lightening) or
- mole that has become symptomatic (pruritic, painful or bleeding)

Clinical features:
- Distribution:
  -- Melanoma occurs as a solitary lesion, and can occur anywhere on the skin.
  -- Most common location → in men: Back / in women: Legs.

Types:
1. Superficial spreading
2. Nodular
3. Acral lentiginous
4. Lentigo maligna

Management:
The first step is to confirm diagnosis histopathologically by excisional biopsy (removing entire lesion with narrow margins and depth through S.C fat). Excisional biopsy with narrow margins is preferred because this allows confirmation of the diagnosis as:
- Pigmented BCC
- Some seborrheic keratoses
- A typical nevi → can mimic melanoma clinically.

Additionally, a complete excision allows determination of:
- Tumor depth (Breslow depth), ulceration, presence of mitosis, regression, lymphatic and vascular involvement and host response.

Excision with wider margins is NOT recommended until the diagnosis is confirmed as:
- it would be inappropriate to remove margins around a benign lesion,
- because this may disrupt afferent cutaneous lymph flow and the ability to identify sentinel nodes.

Primary Prevention:
- Incidence of malignant melanoma has been increasing dramatically → The risk factors for developing melanoma are both:
  1. Environmental: Excessive sun exposure, tanning beds, PUVa therapy, multiple typical/congenital nevi, immunosuppression
  2. Genetic: Caucasian and having a hair complexion, blond hair and blue eyes / Personal history & family history.

- Reducing sun exposure is therefore the best way to prevent the development of melanoma, and
- Use of protective clothing (e.g. long sleeved shirts, wide-brimmed hats, sunglasses, tightly woven fabrics) is one of the most effective methods.

Regarding sunscreens:
- Sunscreens are useful adjuncts to photo-protection, but offer insufficient protection from ultraviolet radiation (UVR) when used alone.
- Little to no protection against melanoma with the use of sunscreen lotion with SPF 15 - 30 → However, there is evidence that SPF protects against non-melanoma skin cancers such as squamous cell carcinoma.
- Overdependence on sunscreens may sometimes even increase risk encourage outdoor exposure.
- Sunscreens should be applied 15-200 min prior to sun exposure to allow enough time for protective film development.

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**Benign Neoplasms & Hyperplasia**

### Seborrheic Keratosis

*Verruca Seborrhoica*

- **Complaint:** Evolve over months to years
  - Are most commonly asymptomatic but can be pruritic or tender, particularly in locations that come into contact with clothing or jewelry.
- **Clinical manifestations:**
  - The natural history is slow enlargement with increasing thickness.
- **Distribution:**
  - Occur in many locations on the body but tend to favor the face & trunk.
  - Lesions DO NOT occur on the palms & soles.
- **Description:** Lesions have a characteristic waxy, "stuck on," warty, and well-circumscribed appearance.
  - Early: Small, 1- to 3-mm, barely elevated [papule](#), later a larger [plaque](#) with or without pigment.
  - Lates: Plaque with warty surface and "stuck on" appearance.
- **Diagnosis:** is based mainly on clinical appearance. Biopsy is rarely required in difficult cases.
- **Differential Diagnosis:**
  - 1. Squamous cell carcinoma (SCC) or basal cell carcinoma (BCC) or malignant melanoma (MM) or Kaposi's sarcoma (KS) (but thrombosed capillaries are present in verrucae).
  - 2. Skin tags.
  - 4. Acne vulgaris.
  - 5. Spreading pigmented actinic keratosis.
- **Treatment:** No therapy is required unless 1. Lesions become irritated or 2. Patient desire for cosmetic reasons. Treatment options are based by agar/steam evaporation or cryosurgery.

### Sebaceous Hyperplasia

- Lesions are 1 to 3 mm and have both telangiectasia & central umbilication.
- **Differential Diagnosis:** This lesion is confused with PASCHER'S TUMOR.
  - PASCHER'S TUMOR is a benign sebaceous hyperplasia that occurs as a result of granuloma formation from infected oil (fatty material) from a sebaceous gland.
- **Treatment:** Destroyed with light electrocautery.

### Lipoma

- Lipomas are single or multiple, benign subcutaneous tumors.
- **Description:**
  - Size: Small BUT may also enlarge to > 6 cm.
  - They are easily recognized because they are soft, rounded, or lobulated and movable against the overlying skin.
  - They occur especially on the neck, trunk, extremities BUT can occur anywhere on the body.
  - **Dimple sign:** lateral compression with thumb and index finger produces a depression or "dimple" in the lesion.
- **Histopathology:**
  - Lipomas are composed of fat cells that have the same morphology as normal fat cells within a C.T framework.
  - Angiolipomas have a vascular component and may be tender in 1. Cold ambient temperature & 2. with compression.
- **Treatment:**
  - Lipomas: should be excised only when considered disfiguring:
    1. Surgical excision
    2. Liposuction can also be performed when lipomas are soft and thus have only a minor C.T component
  - Angiolipomas often require excision

---

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![MedicoNotes](https://www.mediconotes.com)
Vascular Malformation & Tumors

Vascular Tumors (Hemangiomas)

<table>
<thead>
<tr>
<th>Presence at birth</th>
<th>Vascular Malformations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not present at birth but appear postnatally</td>
<td>The are errors of morphogenesis and are presumed to occur during intrauterine life \Most present at birth</td>
</tr>
</tbody>
</table>

| Natural history | | Proportionate growth; can expand |
|-----------------|--------------------------|
| Phases 1: Proliferating; grows rapidly during first year | | Normal endothelial turnover |
| 2: Involuting; slow during childhood over 2 – 6 years. | | |
| 3: Involuting; by the age of 10 years. | | |

<table>
<thead>
<tr>
<th>Cellular</th>
<th>Skeletal changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endothelial hyperplasia</td>
<td>- Occasional mass effect on adjacent bone;</td>
</tr>
<tr>
<td></td>
<td>- rare hyper trophy</td>
</tr>
</tbody>
</table>

Vascular Malformations

<table>
<thead>
<tr>
<th>Hemangioma of infancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Most common tumor of infancy, with F: M ratio ( 3:1 )</td>
</tr>
<tr>
<td>- Clinical Features:</td>
</tr>
<tr>
<td>Description:</td>
</tr>
<tr>
<td>Distribution:</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Syndromic CM:</td>
</tr>
<tr>
<td>Sturge-Weber syndrome (SWS) is the association of Port-Wine Stain in the trigeminal distribution with:</td>
</tr>
<tr>
<td>1: Eye: Vascular malformations in the eye AND 2: Brain: Leptomeninges &amp; superficial calcifications of the brain</td>
</tr>
<tr>
<td>Klippel-Trenaunay-Weber syndrome</td>
</tr>
<tr>
<td>- May have an associated PWS overlying the deeper vascular malformation of soft tissue &amp; bone.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Port-Wine Stain (Nevus flammeus)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Clinical Features:</td>
</tr>
<tr>
<td>Description:</td>
</tr>
<tr>
<td>Distribution:</td>
</tr>
<tr>
<td>- Most commonly involve the face in the distribution of the trigeminal nerve, usually the superior &amp; middle branches</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Syndromic CM:</td>
</tr>
<tr>
<td>Sturge-Weber syndrome (SWS) is the association of Port-Wine Stain in the trigeminal distribution with:</td>
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<td>1: Eye: Vascular malformations in the eye AND 2: Brain: Leptomeninges &amp; superficial calcifications of the brain</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Spidar Angioma (Spider Nevus)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- It consists of a bright red central papule (dilated central arteriole) surrounded by telangiectatic network of dilated capillaries.</td>
</tr>
<tr>
<td>- It’s associated with hyperestrogenic states, such as: pregnancy, in patients receiving estrogen therapy, e.g., oral contraceptives, or in those with hepatocellular disease.</td>
</tr>
<tr>
<td>- Spider angioma may regress spontaneously.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cherry Angioma (Cherry Hemangiomas)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- It appears during third or fourth decade of life</td>
</tr>
<tr>
<td>- They do not regress spontaneously and, because their number often increases with age.</td>
</tr>
<tr>
<td>- They are always cutaneous and NOT found on the mucosa or deep tissues.</td>
</tr>
<tr>
<td>- Description:</td>
</tr>
<tr>
<td>sharply circumscribed areas of congested capillaries &amp; post-capillary venules in papillary dermis.</td>
</tr>
</tbody>
</table>

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### Stevens Johnson syndrome (SJS)

**General Findings:**
- Fever, mentally alert. Distress due to severe pain.
- Cardiovascular: pulse may be >120 beats/min. Blood pressure.
- Renal: tubular necrosis may occur. Acute renal failure.

**Clinical Manifestations:**
- Prodrome: Acute-influenza like (fever, malaise, arthralgias) 1–3 days before mucocutaneous lesion.
- Skin lesions:
  - **Prodromal Rash:** Morbilliform, can be target lesion-like eruption
  - **Necrosis & sloughing of epidermis:** (exfoliation of the skin)
    - First appears as macular areas with crinkled surface that enlarge and coalesce.
    - **Sheetlike loss of epidermis.**
    - **Raised flaccid blisters that spread with lateral pressure (Nikolsky sign)** on erythematous areas.
- **Mucous membranes:**
  - There is typically a sudden onset of mucocutaneous lesion over two sites usually:
    1. **Oral:** Erythema, painful erosions & blisters
    2. **Conjunctivitis:** 85% have conjunctival lesions
      - Hyperemia, pseudomembrane formation; keratitis, corneal erosions; later synechiae between eyelids and bulbar conjunctiva.
- **Systemic:**
  - Fever, malaise, arthralgia, myalgia, lymphadenopathy, rash, angioedema, nausea, vomiting, diarrhea.
  - **Renal:** Acute renal failure.
  - **Cardiovascular:** Ascites, pericardial effusion, heart failure, hypotension, pleural effusion.
  - **Hematologic:** Anemia, leukocytosis, leukopenia, thrombocytopenia, coagulopathy.
  - **Others:** Acute respiratory distress syndrome, toxic shock syndrome, sepsis, septic shock.

**Pathogenesis:**
- Drugs or their metabolites act as haptens and render keratinocytes antigenic by binding to their surfaces → cytotoxic immune reaction aimed at the destruction of keratinocytes expressing foreign (drug-related) antigens.
- Epidermal injury is based on the induction of apoptosis.

**Serious Adverse Cutaneous Drug Reactions (SADRs)**

- **Type I (Immediate)**: IgE-mediated reaction
  - Immediate type immunologic reactions:
    - Some patients can form drug-specific (IgE) on exposure to a medication → upon drug re-exposure IgE binds to basophils & mast cells → Symptoms.
  - **Symptoms:** Urticaria, pruritus, flushing, asthma, bronchial constriction, hypotension, anaphylaxis.

- **Type II (Cell-mediated)***
  - Drug + cytotoxic antibodies cause lysis of cells such as platelets or leukocytes
  - IgG or IgM antibodies formed to drug; immune complexes deposited in small vessels activate complement and recruitment of granulocytes

- **Type III (Immune complex)***
  - Immune complex reaction
  - Hypersensitivity reaction caused by drug, liberating cytokines, which trigger cutaneous inflammatory response

- **Type IV (Tumor-antigen-antibody)***
  - Cell-mediated immune reaction
  - Sensitized lymphocytes react with drug, liberating cytokines, which trigger cutaneous inflammatory response

**Clinical Findings**

<table>
<thead>
<tr>
<th>Type I</th>
<th>Type II</th>
<th>Type III</th>
<th>Type IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgE-mediated: immediate-type immunologic reactions:</td>
<td>Drug + cytotoxic antibodies cause lysis of cells such as platelets or leukocytes</td>
<td>IgG or IgM antibodies formed to drug; immune complexes deposited in small vessels activate complement and recruitment of granulocytes</td>
<td>Cell-mediated immune reaction: Sensitized lymphocytes react with drug, liberating cytokines, which trigger cutaneous inflammatory response</td>
</tr>
<tr>
<td>Onset is rapid (seconds to minutes)</td>
<td>Immunoglobulins</td>
<td>Sulfamethoxazole,</td>
<td></td>
</tr>
<tr>
<td>Prodrome: Acute-influenza like (fever, malaise, arthralgias) 1–3 days before mucocutaneous lesion</td>
<td>2-Antibiotics</td>
<td>Anticonvulsants</td>
<td>Anticonvulsants</td>
</tr>
<tr>
<td>Skin lesions: Prodromal Rash: Morbilliform, can be target lesion-like eruption</td>
<td>3-Rituximab</td>
<td>3-Allopurinol</td>
<td>3-Allopurinol</td>
</tr>
<tr>
<td>Necrosis &amp; sloughing of epidermis: (exfoliation of the skin)</td>
<td>4-Infliximab</td>
<td>4-Infliximab</td>
<td>4-Infliximab</td>
</tr>
</tbody>
</table>

**Common triggers**

- Drugs
  - Allopurinol
  - Antibiotics (eg, sulfonamides)
  - Anticonvulsants (eg, carbamazepine, lamotrigine, phenytoin)
  - NSAIDs (eg, piroxicam)
  - Sulfasalazine
  - Other
  - Mycoplasma pneumoniae
  - Vaccination
  - Graft-vs-host disease

**Immunologically Mediated A Cutaneous Drug Reactions**

- SJS and TEN are different from Erythema Multiforme (EM).
- New consensus that SJS and TEN are different from Erythema Multiforme (EM).
- Onset is rapid (seconds to minutes).
- Prodrome: Acute-influenza like (fever, malaise, arthralgias) 1–3 days before mucocutaneous lesion.
- Skin lesions: Prodromal Rash: Morbilliform, can be target lesion-like eruption
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  - Renal: tubular necrosis may occur. Acute renal failure.

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Erythema Multiforme
- A common reaction pattern (to a variety of antigenic stimuli) of blood vessels in the dermis with secondary epidermal changes.
- Reaction is most commonly to Herpes Simplex Virus (HSV) Infection.

Antigenic stimuli:
- Infection: Especially following herpes simplex, Mycoplasma.
- Drugs: Sulfonamides, phenytoin, barbiturates, phenylbutazone, penicillin, allopurinol.
- Idiopathic: Probably also due to undetected herpes simplex or Mycoplasma.

Clinical Features:
- Description:
  - Dull red iridescent or target-like lesions (sharply defined flat papules with a central vesicle) are typical.
- Distribution:
  - Bilateral and often symmetrical.
  - Either: 1. Localized to hands & face or 2. Generalized: Bilateral and often symmetric.

Mucous Membranes: Erosions with fleshy membranes, occasionally ulcerations.
- lips, oropharynx, nasopharynx, vulvar, anal.

Erythroderma (Exfoliative Dermatitis)
- NO Vesicles
- Little or NO mucous membrane involvement
- NO Nodule

Epidermal reactions are usually associated with an outbreak of herpes simplex (HSV) preceding it by several days.

Complications of HSV Infections
- Eczema herpeticum: Usually follows autoinoculation of HSV (most commonly orolabial herpes) to atopic dermatitis.
- Staphylococcus aureus infection: Occurs with eczema herpeticum.
- Erythema multiforme: In some individuals with recurrent HSV infections, erythema multiforme may occur with each recurrence.

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Differential Diagnosis
- It's a severe, life-threatening reaction pattern of the skin characterized by generalized and uniform REDNESS and SCALING involving practically the entire skin.
- 50% of patients have history of preexisting dermatosis: In the order of frequency: Psoriasis, atopic dermatitis, adverse cutaneous drug reactions, cutaneous T cell lymphoma, allergic contact dermatitis, and pityriasis rubra pilaris.
- It is associated with fever, malaise, shivers, and generalized lymphadenopathy.
- There is a loss of scalp and body hair, and nails separated from nail bed (onycholysis).
- There may be hyperpigmentation in whose normal skin color is brown or black.

Erythema Induratum (Nodular Vasculitis)
- Nodular vasculitis: A form of lobular panniculitis associated with subcutaneous blood vessel vasculitis with subsequent ischemic changes that produce lipocyte injury, necrosis, inflammation, and granulation.
- Appears as a nodular eruption in patients with Tuberculosis.
- On examination:
  - There are crops of small, tender, erythematous nodules involving the shins and calves: may undergo necrosis forming slowly healing ulcers.

Erythema Nodosum
- The most common type of panniculitis:
  - Panniculitis: the term used to describe diseases where the major focus of inflammation is in the S.C tissue.
- EN is an important and common acute inflammatory/immunologic reaction pattern of the subcutaneous fat.
- Characterized by: Appearance of painful nodules on the lower legs.
  - Lesions are bright red and flat but nodular upon palpation.
- EN is not a disease but a cutaneous reaction pattern to various etiologic agents.
Wariarin-induced skin necrosis:
- It’s a serious complication of oral anticoagulants.
- Protein C deficiency is sometimes associated with this condition.
- Females are most commonly affected.

Clinical Features:
- **Distribution:** The commonly involved sites: breasts, buttocks, thighs, and abdomen.
- **Description:** The initial complaint is pain, followed by bullae formation & skin necrosis (It mostly occurs within weeks after starting therapy)

Treatment:
- Vitamin K should be promptly administered in the early stages of the lesion
- Wariarin is discontinued if the lesion progresses & Heparin should be used to maintain anticoagulation until necrotic lesions heal.
- Skin grafting: Few patients require it.

**Urticaria & Angioedema**

Urticaria:
- Composed of wheals (transient edematous papules and plaques, usually pruritic and due to edema of the papillary body).
- Superficial, well defined.

Angioedema:
- Larger edematous area that involves the dermis and subcutaneous tissue.
- Deep and ill defined.

**Urticaria and angioedema are the same edematous process but involving different levels of the cutaneous vascular plexus.**

Acute urticaria:
- Acute onset and recurring over <30 days.
- Usually large wheals often associated with angioedema.
- Etiology:
  - Often IgE-dependent with atopic diathesis.
  - Related to foods, parasites, and drugs (penicillin).
- Description:
  - Sharply defined round/oval wheals, small (<1 cm) to large (>8 cm), erythematous.
  - Clearing of the central region may occur (white with an erythematous rim).
  - Lesions may coalesce, producing an annular, serpiginous pattern.
  - Pruritus → in angioedema of palms and soles pain.

Acute urticaria & angioedema:
- Note that there are both:
  1. Superficial wheals &
  2. Deep, diffuse edema.

Angioedema:
- Skin-colored, transient enlargement of portion of face, eyelids, lips, tongue, glottis & larynx, or other sites due to subcutaneous edema.
- Laryngeal edema can cause airway obstruction and be life threatening.

ACE inhibitors + Angioedema:
- ACE inhibitors are the most common cause of acquired angioedema.
- **Mechanism:**
  - ACE is also known as kininase, it functions to degrade bradykinin.
  - When ACE is inhibited → levels of bradykinin increase → thereby leading to angioedema.
  
  Angioedema occurs due to the pro-inflammatory action of bradykinin, which promotes edema, inflammation and the sensation of pain.

  - It is important to note that angioedema from ACE inhibitors can occur at ANYTIME, not just within weeks of starting the medication.
- **Treatment:** (ACE-inhibitor should be stopped immediately)
  - First step in management of angioedema is: to check for 1. Airway compromise & 2. Vasomotor instability, → which require S.C epinephrine administration if present.
  - If airway obstruction fails to respond to epinephrine → emergency tracheostomy is done.

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Hereditary Angioedema (HAE)

- A serious autosomal dominant disorder, may follow trauma (physical and emotional).

- Pathophysiology:
  - C1-esterase inhibitor deficiency, dysfunction or destruction
    - Since C1-esterase inhibitor is also the major inhibitor of the Hageman factor & kallikrein, the two enzymes required for kinin formation → elevated levels of the edema-producing factors C2b & bradykinin
  - Episodes usually follow an infection, dental procedure or trauma.

- Clinical Manifestations:
  - Usually presents in late childhood
  - It is characterized by a rapid onset of the following symptoms:
    - (1) non-inflammatory edema of the face, limbs, genitalia,
    - (2) laryngeal edema, and
    - (3) edema of the intestine, resulting in colicky abdominal pain.

- Laboratory Abnormalities:
  - C1-esterase inhibitor: Decreased levels of (85%) / dysfunctional inhibitor (15%),
  - C1q levels: Normal in hereditary angioedema (depressed in acquired forms)
  - C4 levels: Abnormal in all forms of angioedema.

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Vitiligo

- It's a depigmenting disorder due to loss of epidermal melanocytes.
- Pathogenesis: it's thought to be an autoimmune process which causes destruction of the melanocytes. Progressive, acquired, chalk-white, bilateral (usually symmetric).

- Associations:
  Vitiligo is associated with other autoimmune conditions like: pernicious anemia, autoimmune thyroid disease (usually Graves' or chronic autoimmune thyroiditis), type 1 diabetes mellitus, primary adrenal insufficiency, hypopituitarism, and alopecia areata

- Clinical Features:
  Description: progressive, acquired, chalk-white, bilateral (usually symmetric), sharply marginated macules with hyperpigmented borders in typical sites.
  Distribution:
  - Localized type: characterized by one or several macules in a single site:
    - is more common and is characterized by widespread distribution of depigmented macules, often in a remarkable symmetry as on the low back and in genital areas
  - Generalized vitiligo:
    - Systemic photochemotherapy (oral PUVA)
    - Narrow-band ultraviolet light B (UVB, 311 nm) is now the treatment of choice
    - Excimer laser (308 nm):
      - Produces best results in the face.
  - Depigmentation:
    - Bleaching: Bleaching of normally pigmented skin with monobenzylether of hydroquinone (MEH) cream is a permanent, irreversible process.

Alopecia

- Alopecia Areata
  - A localized loss of hair in round or oval areas with no apparent inflammation of the skin
  - Pathogenesis:
    - Follicular damage occurs in anagen followed by rapid transformation to catagen and to telogen, then to dystrophic anagen status.
    - While the disease is active, follicles unable to progress beyond early anagen and do not develop normal hair.
  - Clinical Features:
    Description: Skin is Normal
    Round patches of hair loss.
    - Single or multiple. May coalesce.
    - It's often sharply defined.
    - Normal-appearing skin with follicular openings present.
    - "Exclamation mark" hairs: Diagnostic broken-off stubby hairs (distal ends are broader than proximal ends) seen at margins of hair loss areas.
  - Distribution:
    - Most commonly on scalp
    - Any hair-bearing area: Beard, eyebrows, eyelashes, pubic hair.
  - Management: No curative treatment is currently available. Treatment for AA is unsatisfactory.
    Treatment directed at inflammatory infiltrate and growth inhibitor factors produced by inflammation.
    1. Glucocorticoids: Glucocorticoids Topical / Intralosional injection / Systemic Glucocorticoids
    2. Systemic Cyclosporine: Induces regrowth, but AA recurs when drug is discontinued.
    3. Induction of Allergic Contact Dermatitis: By Dinitrochlorobenzene (DNCP), but local discomfort.
    4. Oral PUVA (Photochemotherapy): Entire body must be exposed.
# Psoriasis

- **Classification:**
  1. Psoriasis vulgaris:
     - Acute guttate
     - Chronic stable plaque
  2. Psoriatic erythroderma
  3. Pustular psoriasis
  4. Psoriasis arthropitis

- **Triggering Factors:**
  - External: Rubbing & scratching stimulate the psoriatic proliferative process. Occurs 7 to 14 days after injury.
  - Internal: Systemic triggering factors and Koebner (Wickham striae)

- **Distribution:**
  - Sites: Scalp, Most obvious at the hair line & intergluteal cleft, behind the ears.
  - Form of presentation: Single lesion or lesions. Can be grouped, annular, or disseminated scattered discrete lesions when generalized.

- **Clinical Features:**
  - Lesions have been designated as the four P’s: Sharply marginated, raised red plaque with a white scale surface that coalesce to form geographic lesions. Lesions can vary in size from papule to plaque. Scales are lamellar, loose, and easily removed by scratching.

- **Symptoms:**
  - Asymptomatic or pruritic. Onset: Acute (days) or insidious (over weeks).

- **Treatment:**
  1. **Systemic Therapy:**
     - Glucocorticoids: topical WITH occlusion for cutaneous lesions.
     - Intravenous is helpful for:
       - Symptomatic cutaneous or oral mucosal lesions and lips.
  2. **Topical Therapy:**
     - Calcipotriene, Tazarotene, Calcipotriol
     - Steroids: Emollients, Aquaphor, and Vaseline or mineral oil.

  - Cyclosporine and Tacrolimus Solutions
  - Ultraviolet light
  - Methotrexate

- **Referral:** In dark-skinned individuals, postinflammatory hyperpigmentation.

---

# Lichen Planus

- **Description:**
  - Characterized by flat-topped, pink to violaceous, with white lines (Wickham striae), shiny, pruritic, polygonal PAPULES.

- **Distribution:**
  - Mucous membranes of the cheeks & lips: Common on the flexor surfaces of wrists, the forearm & legs: On intertriginous areas of flexion.

- **Clinical features:**
  - Lesions are purple, polygonal, or papular. In dark-skinned individuals, postinflammatory hyperpigmentation.

- **Treatment:**
  1. **Local Therapy:**
     - Glucocorticoids
  2. **Systemic Therapy:**
     - Cyclosporine
     - Methotrexate, in very resistant and generalized cases.
  3. **PUVA Photochemotherapy:**
     - Retention “mouthwash” for severely symptomatic oral LP.

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Acanthosis Nigricans (AN)

- A cutaneous marker related to hereditary, obesity, endocrine disorders (particularly diabetes), drug administration & malignancy.
- Clinical features:
  - Description: hyperkeratotic, hyperpigmented plaques with a classic "velvety" texture.
  - Skin tags (acrochordons), which are pedunculated outgrowths of normal skin, are also commonly present on regions affected by AN → figure
  - Distribution: Flexural areas: axilla, groin, posterior neck, are the most common locations affected.
- Depending on the etiology, AN can be divided into benign and malignant forms:

<table>
<thead>
<tr>
<th>Benign AN</th>
<th>Malignant AN</th>
</tr>
</thead>
<tbody>
<tr>
<td>It's typically seen in younger individuals.</td>
<td>It is associated with underlying neoplasms, especially of the</td>
</tr>
<tr>
<td>The sudden appearance of such skin changes in middle-aged or elderly</td>
<td>gastrointestinal tract and genitourinary tract</td>
</tr>
<tr>
<td>patients is suggestive of underlying malignancy.</td>
<td>Lesions can occur in uncommon areas (e.g., mucous membranes, palms, soles)</td>
</tr>
<tr>
<td>It is associated with insulin-resistant states</td>
<td></td>
</tr>
<tr>
<td>The skin is usually dry and rough with horny plate over the extensor</td>
<td></td>
</tr>
<tr>
<td>surfaces of the limbs.</td>
<td></td>
</tr>
<tr>
<td>In children, there may be relative sparing of the face and diaper area.</td>
<td></td>
</tr>
<tr>
<td>The condition worsens in the winter because of increased dryness, and sometimes referred to as &quot;lizard skin.&quot;</td>
<td></td>
</tr>
</tbody>
</table>

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