

DIAGNOSIS & MANAGEMENT OF Dermatological DISORDERS

In Skin Infections



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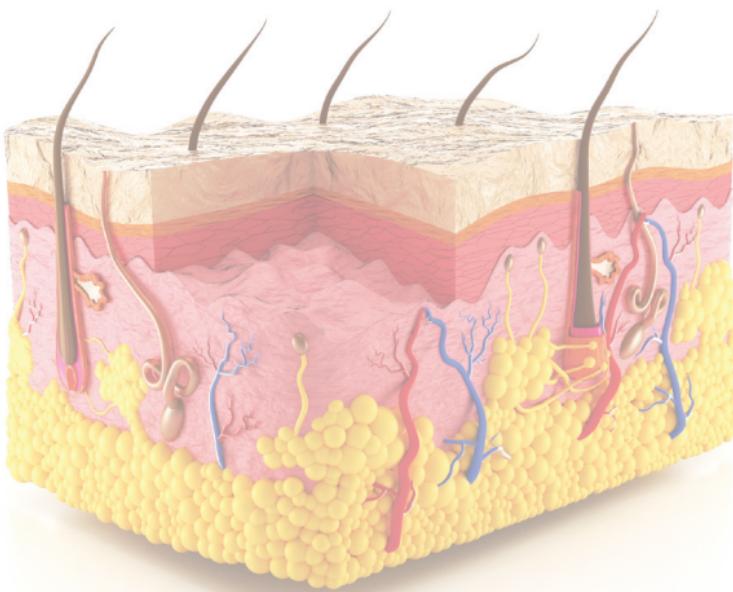
SKINNOVA

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PREFACE

Skin problems often pose a challenge for the practicing physicians because of their frequently overlapping clinical presentations, and a myriad of terms that are often used interchangeably. Oftentimes, a skin disorder may be presenting feature of some underlying systemic problem also. It is therefore important that physicians are aware of the common terms used in dermatology practice.

This book is aimed to provide a condensed, organized and practical approach towards patients presenting with common skin problems; and is intended to be equally useful for medical students, primary care physicians, and dermatologists. Each section of the book covers some of the most common skin diseases seen in dermatology practice, navigating the readers through elements of typical clinical presentations, diagnostic features, and treatment options available for these conditions.



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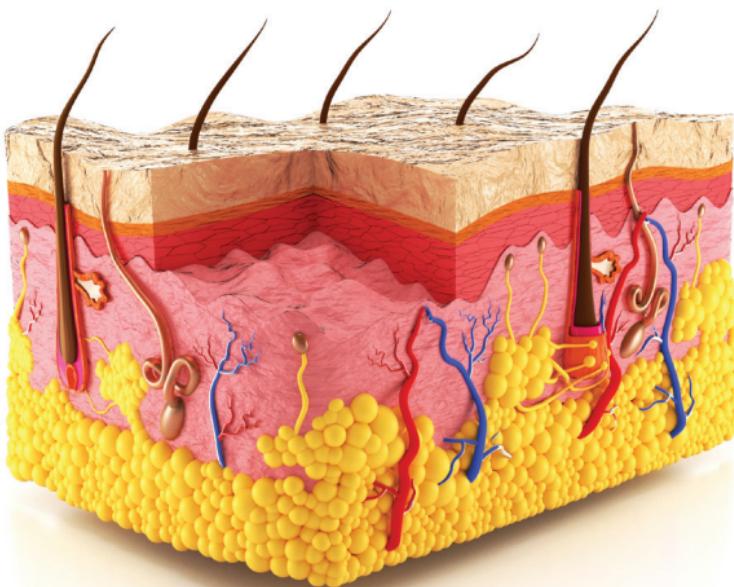
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SECTION 1

THE BIOLOGY OF SKIN & ITS APPENDAGES

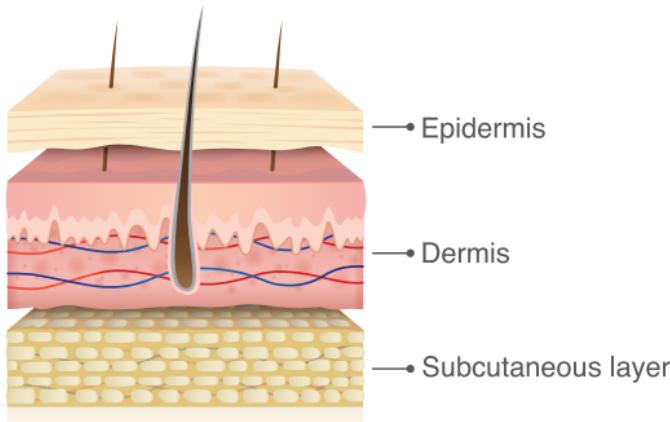


■ ANATOMY OF SKIN

Skin is the outermost and largest organ of the body that covers body's entire external surface, and is often viewed as an envelope and barrier to protect the body from the outside world. Internally, it is closely integrated to the underlying fascial endoskeleton through various tissues like retinacular ligaments, blood vessels, nerves and lymphatics.

Anatomically, the human skin is composed of three distinct layers - epidermis, dermis and hypodermis, all of which vary significantly in their structure and function and the degree of specialization within each layer. Furthermore, the thickness of each layer varies depending on the body region.

Three main layers of the skin



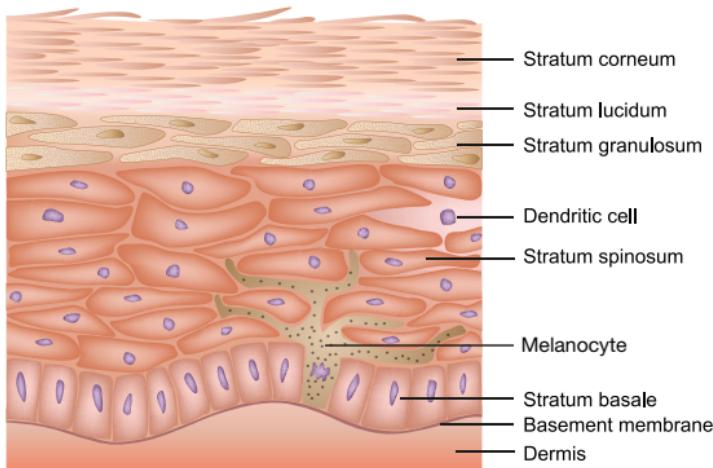
■ EPIDERMIS

Epidermis is the most superficial and biologically active of skin layers since the basal layer of epithelium (Stratum basale) is constantly

renewing. The layer – epidermis – is densely packed by epithelial cells to a depth of between 75 and 150 µm (up to 600 µm thick on palms/soles). Different layers of the epidermis include:

1. Stratum corneum (the most superficial portion of the epidermis)
2. Stratum lucidum
3. Stratum granulosum
4. Stratum spinosum
5. Stratum basale (the deepest portion of the epidermis; also known as stratum germinativum)

Structure of the epidermis



Structurally, a transition is seen in the epidermis, with a nuclear cells of stratum corneum present superficially to distinct hexagonal shaped cells present in the stratum basale. The basement membrane forms an important part of dermo-epidermal junction that adheres epidermis with dermis, and provides a strong mechanical barrier.

■ CHARACTERISTICS OF DIFFERENT LAYERS OF EPIDERMIS

Stratum corneum – the uppermost layer of epidermis

- 20-30 cell layers. Made up of keratin and horny scales made up of dead keratinocytes, known as anucleate squamous cells
- Varies most in thickness, especially in callused skin. Contains dead keratinocytes, which secrete defensins (part of our first immune defense).

Stratum lucidum

- 2-3 cell layers. Present in thicker skin found in palms and soles
- A thin clear layer consisting of eleidin, a transformation product of keratohyalin.

Stratum granulosum

- 3-5 cell layers
- Contains diamond shaped cells with keratohyalin granules and lamellar granules. Keratohyalin granules contain keratin precursors, which eventually aggregate, crosslink, and form bundles. Lamellar granules contain glycolipids that get secreted to cells' surface and function as a glue to keep the cells stuck together.

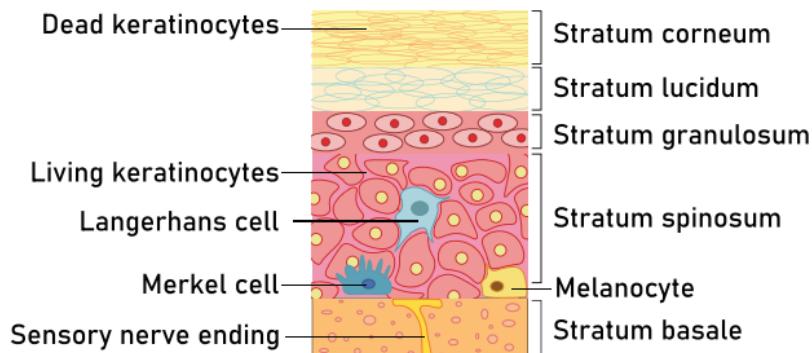
Stratum spinosum – also known as the prickle cell layer

- 8-10 cell layers
- Contains irregular, polyhedral cells with cytoplasmic processes - sometimes called "spines", which extend outward and contact neighboring cells by desmosomes.
- Dendritic cells can be found here.

Stratum basale – also known as stratum germinativum

- The deepest layer of epidermis
- Separated from the dermis by basement membrane (basal lamina)
- Attached to basement membrane by hemidesmosomes
- Contains cuboidal to columnar mitotically active stem cells, which constantly produce keratinocytes
- Also contains melanocytes.

Layers and cells of epidermis



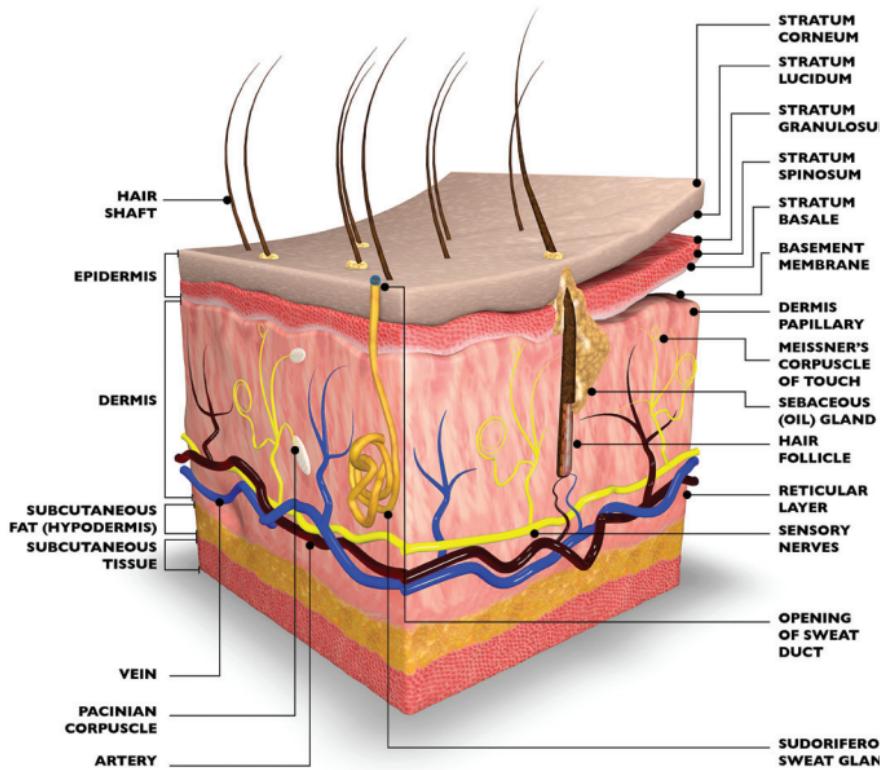
Cells of epidermis

- Keratinocytes
- Melanocytes
- Langerhans' cells
- Merkel's cell

■ DERMIS

The dermis is usually less than 2 mm thick, but may be up to 4 mm thick in some individuals, and is known to provide most of the mechanical strength to the skin. The layer consists of two regionally distinct layers of connective tissue, which merge together without clear demarcation:

Dermis



- (i) The superficial and thinner papillary dermis, which interacts closely with epidermal rete ridge projections and surrounding individual hair follicles
- (ii) The thicker and deeper reticular dermis, which consists of connective tissue/bundles of collagen fibers. This layer is made up of predominantly large diameter collagen fibres, which is less densely packed and organized into large inter-woven fibre bundles of branching elastic fibres.

Dermis also houses sweat glands, hair follicles, muscles, sensory neurons, and blood vessels.

■ HYPODERMIS

Hypodermis - also called subcutaneous fascia - is the deepest layer of skin and mainly consists of loose connective tissue that insulates the skin. The layer contains adipose lobules together with some skin appendages like hair follicles, sensory neurons, and blood vessels.

Cells found in hypodermis include:

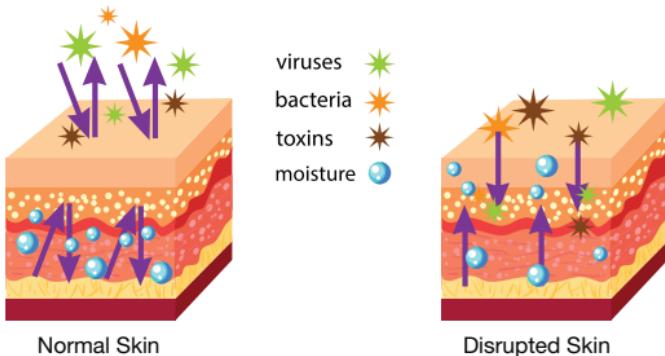
- Fibroblasts
- Adipose cells
- Macrophages.

■ FUNCTIONS OF SKIN

Skin has many functions

- a. Particularly, the barrier function of the skin is well-known, having significance with regards to several environmental challenges, like pathogens, UV light, and chemicals, and mechanical injury.

Skin barrier function



- b. In addition, skin also tends to provide a 'live feed' of information of the body's systemic physiology through a variety of physical signs, such as flush, sweating and pallor, which can be pointing to some underlying disease states.
- c. It provides sensation to touch, heat, cold, and pain by the actions of the nociceptors.
- c. Skin also regulates temperature and the amount of water released into the environment, thereby preserving body homeostasis.
- d. Skin also serves both endocrine (production of vitamin D) and exocrine functions (by way of the sweat and sebaceous glands).

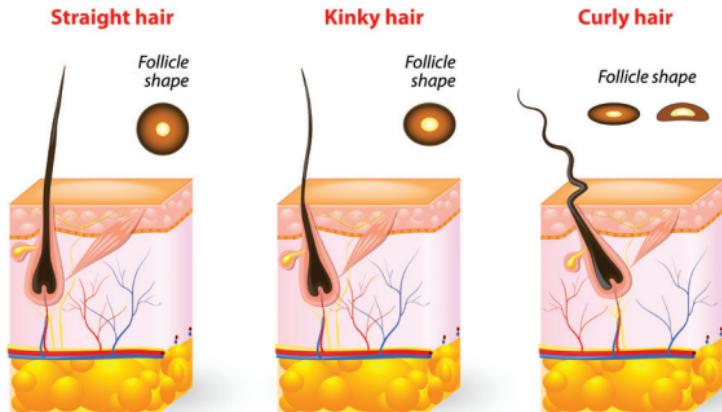
One feature that is often overlooked is the dynamism of skin, which involves multidirectional stretch and compression that allows for low friction gliding movement and is important to daily activity.

■ SKIN APPENDAGES

■ HAIRS

Hairs are characteristic to mammals, which develop in the skin as epidermal down-growths that invade the underlying dermis. In humans, hair grows at a rate of 0.35 mm/day.

Hairs



Anatomy of Hair

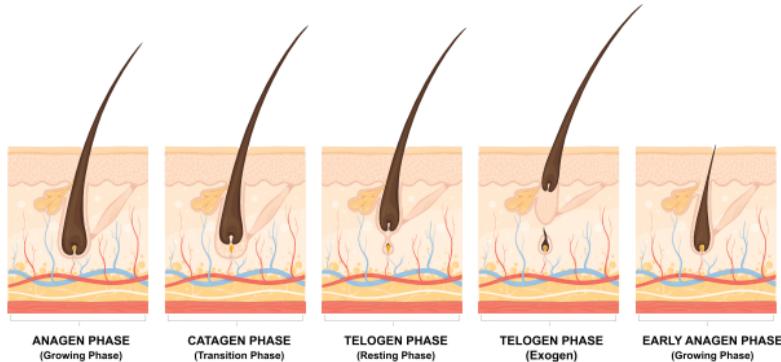
Hair consists of two distinct parts: hair follicle - the living part located under the skin, and the hair shaft. The structural pilosebaceous unit comprises the hair follicle itself, an attached sebaceous gland and arrector pili muscle. The external shaft is consisted of 3 layers: cuticle, cortex, and in certain cases medulla.

SECTION 1

Hair follicles cycle through three primary phases:

- Anagen (or growth phase),
- Catagen (transition or regression phase), and
- Telogen (resting phase).

Hair growth cycle



Anagen phase lasts 2 to 6 years, during which primary hair growth occurs. At any given time, between 85% and 90% of hair follicles are in this phase. Catagen, or transitional, phase is the shortest of all 3 phases (2–3 weeks), and is characterized by follicular regression. Telogen phase lasts approximately 3 months. After these three phases, the inactive (dead) hair is shed. Humans shed around 100 hairs daily.

Functions of Hair

Human hair serves several physiological and psychological functions, including thermoregulation, protection, and individual impact on person's identity, self-image and self-evaluation.

Hair aids in sweating thermoregulation, and help to maintain temperature by retaining heat or preventing cold, while also protecting

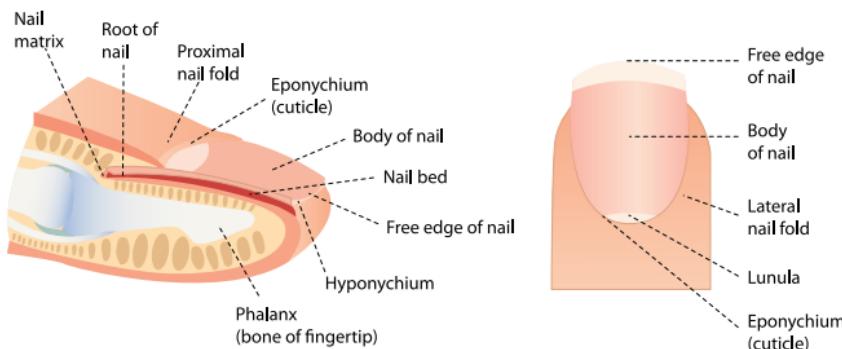
from elements like UV radiation. Humans also have specialized hair, such as eyelash and eyebrow hair.

The psychological function of hair is especially evident in disorders of hair loss.

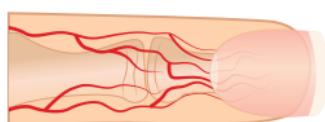
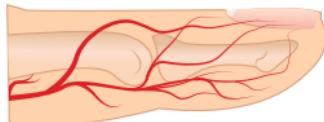
■ NAILS

Nails are hardenings of the horny zone of the epidermis, which overlie the dorsal aspect of distal phalanges. Their growth is affected by nutrition, hormones, and disease, and involves considerable protein synthesis, because of which non-specific changes occur in nails in response to local and systemic disturbances; for e.g., white spots indicate incomplete keratinization.

Nail structure



BLOOD SUPPLY



Anatomy of Nails

The nail unit comprises nail plate, surrounding soft tissues, and their vasculature and innervation based upon the distal phalanx. The nail plate - embedded by the proximal and lateral folds - is a laminated keratinized structure lying on the nail matrix (15-25%), the nail bed with its distal onychodermal band (75-85%), and the hyponychium at its free edge. From the proximal nail fold, the cuticle adheres to the superficial surface of proximal nail plate. Nail plate makes up only 1/3rd of the length of the nail unit, and only the distal portion is visible, as most remains hidden underneath the proximal nail fold. The entire nail unit possesses abundant vascular network to ensure adequate blood supply. The nerves innervating the nail unit mimic the arterial supply.

Function of Nails

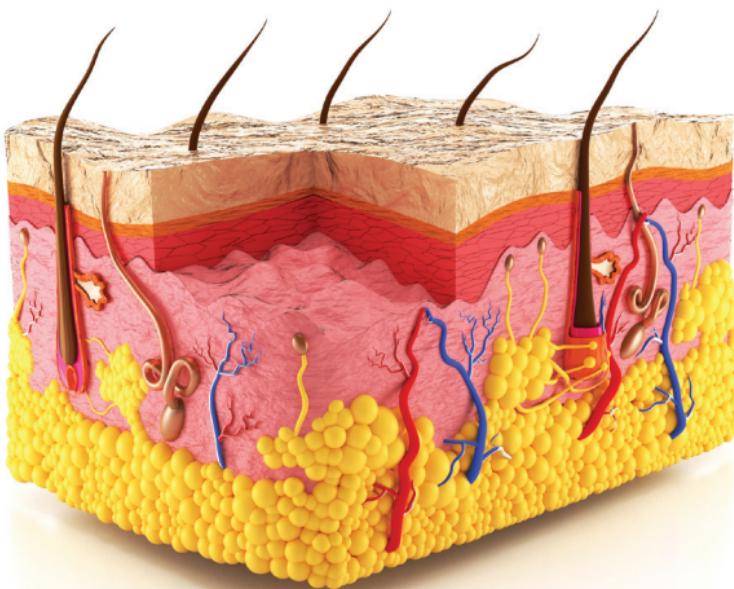
Nails fulfill the general and indispensable function of protecting the terminal phalanx and fingertip from traumatic impact, and contributing to tactile sensation by acting as a counterforce to the fingertip pad. In the fingers, they serve in scratching. In humans, nails are also used frequently for grooming and alleviation of itching than for offense or defense. They may serve a cosmetic purpose through a variety of manipulations and modifications.

Functions of nail unit

- Protection of phalanges and fingertips
- Enhancement of fine touch and fine digital movements
- Scratching and grooming
- Aesthetic and cosmetic purpose

SECTION 2

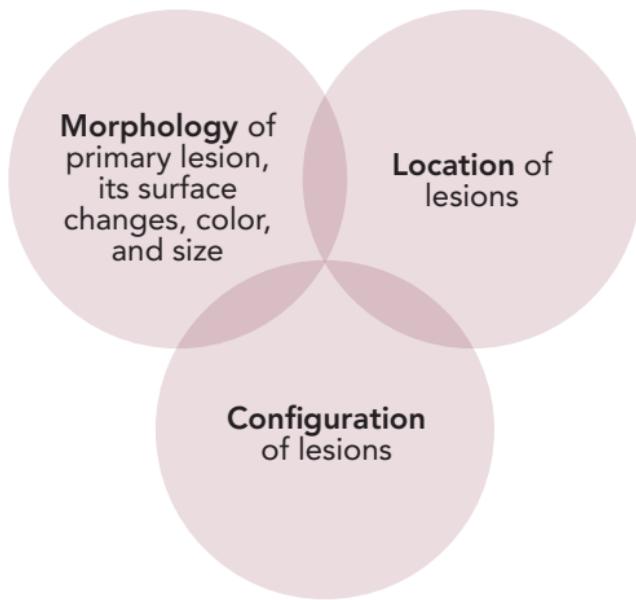
CLINICAL METHODS & COMMON TERMS



■ CLINICAL METHODS IN DERMATOLOGY

In day-to-day dermatology practice, visual pattern recognition is a common method frequently used to make a clinical diagnosis; dependent on accurate and complete description and matching of the cutaneous findings/skin lesions.

Most common features of skin disorders that are used in pattern recognition



The method appears more effective for common skin disorders with typical presentations. Herein, accurate examination helps to distinguish the type of lesions, while palpation can provide additional

information on the lesions' surface and texture. Therefore, at initial physical examination, all cutaneous abnormalities should be noted using appropriate morphologic descriptive terms.

A complete examination should be performed, rather than looking at only what the patient may think is important or suitable for inspection; otherwise, the primary lesion, or a more important lesion of which the patient is unaware, may be missed.

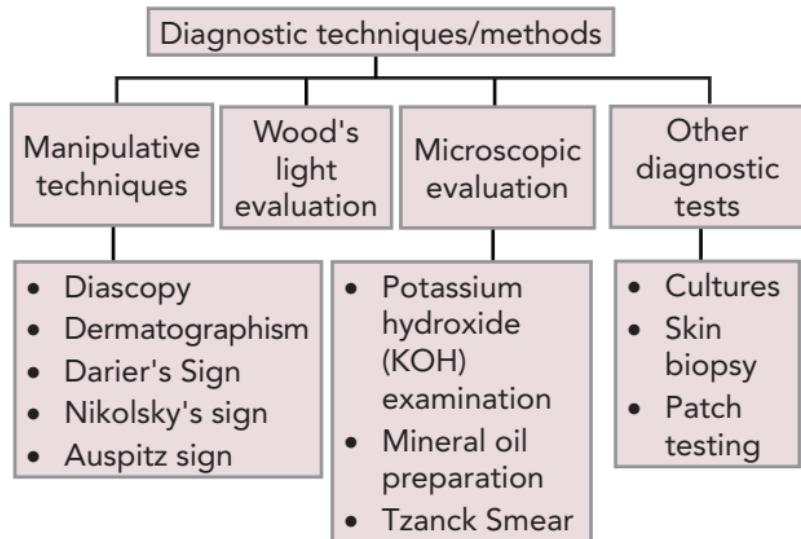
Prior to physical examination, however, clinical history taking by the physician should establish the duration and location of the skin problem, and what change has brought the patient to physician. Concurrently, patient's age, occupation, state of health, and current medications should be also determined.

■ DIAGNOSTIC TECHNIQUES/METHODS

As mentioned earlier, many skin diseases may be quickly recognized with help of their characteristic clinical appearance. However, several simple diagnostic procedures are also available, which often provide a definitive method of differentiating between morphologically similar skin problems, and may help the clinician in confirming his/her initial diagnostic impression.

Additionally, with continued technological advancement, several ancillary imaging technologies are now available to aid in diagnosis and management of skin problems.

Diagnostic techniques/methods



■ IMAGING TECHNOLOGIES IN DERMATOLOGY

- Dermoscopy
- Digital photographic imaging
- Confocal microscopy
- Optical coherence tomography
- High-frequency ultrasound
- Raman spectroscopy
- Fluorescence imaging

■ COMMON TERMS IN DERMATOLOGY

Primary & secondary lesions

Primary lesions represent the morphologic changes most representative of a pathologic process, and the basis of diagnostic categories of dermatologic disease. Several generic nouns can be used to describe a skin lesion based on its primary visual characteristic. Diligent examination is required to discover these primary lesions as they often do not represent the first lesions experienced by the patient. Subsequently, when primary lesions have been altered by disease progression or external factors like overtreatment, excessive scratching, or infection of a primary lesion, secondary changes are seen.

Appropriate adjectives can be added to the primary and secondary terms to evoke an accurate visual descriptive image. It is however important to note that an "accurate image" only implies generic descriptive terminology, and not a diagnosis.

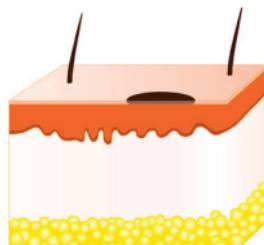
Primary lesions	Secondary changes	Adjectives
<ul style="list-style-type: none"> • Macule • Papule • Plaque • Nodule • Vesicle • Bulla • Pustule • Wheal • Comedo • Burrow • Cyst 	<ul style="list-style-type: none"> • Scale • Crust • Fissure • Erosion • Ulcer • Excoriation • Atrophy • Scar 	<ul style="list-style-type: none"> • Color <ul style="list-style-type: none"> » White » Red » Purple » Brown • Surface <ul style="list-style-type: none"> » Smooth » Rough • Feel <ul style="list-style-type: none"> » Soft, Firm, Tender

■ CHARACTERISTICS OF PRIMARY & SECONDARY LESIONS

A. Primary lesions

1. Macule

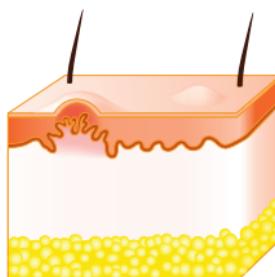
- A flat, circumscribed, nonpalpable lesion that differs in colour from the surrounding skin; can be any colour or shape



Macule

2. Papule

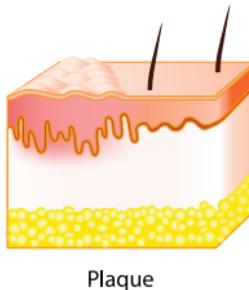
- An elevated, solid, palpable lesion that is ≤ 1 cm in diameter.



Papule

3. Plaque

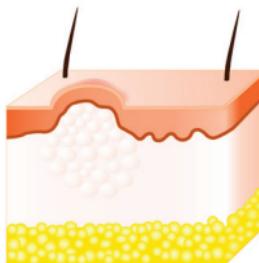
- A circumscribed, palpable lesion > 1 cm in diameter; most plaques are elevated. Plaques may result from a coalescence of papules.



Plaque

3. Nodule

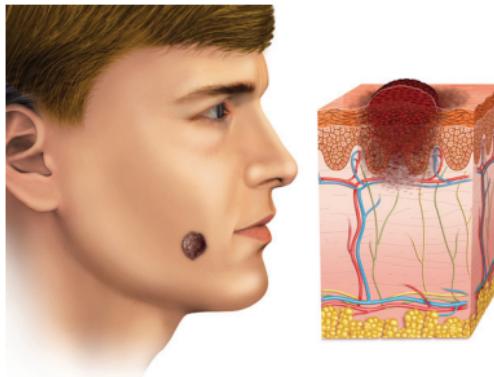
- An elevated, solid, palpable lesion > 1 cm usually located primarily in dermis and/or subcutis. The major portion of the lesion may be beneath the skin surface.



Nodule

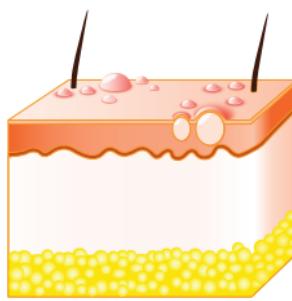
4. Tumor

- A large nodule; a nodule $>$ 2 or 3 cm in diameter is usually called a tumor.



5. Vesicle (blister)

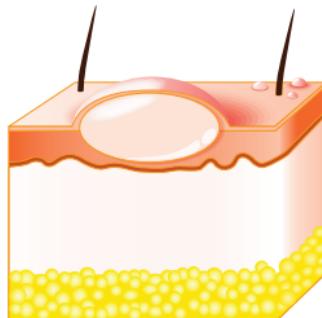
- A circumscribed lesion \leq 1 cm in diameter that contains liquid (clear, serous or hemorrhagic).



Vesicle

6. Bulla

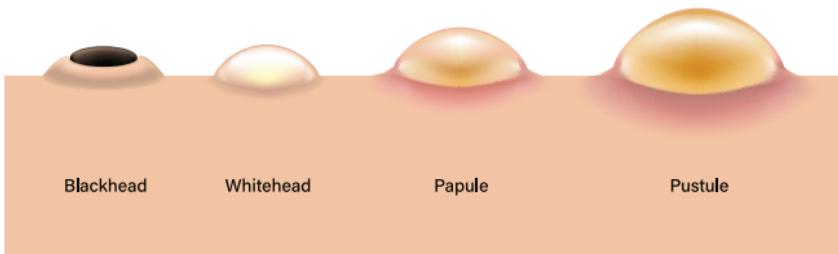
- A larger vesicle; >1 cm, that contains liquid (clear, serous or haemorrhagic).



Bulla

7. Pustule

- A circumscribed lesion that contains pus.



8. Wheal

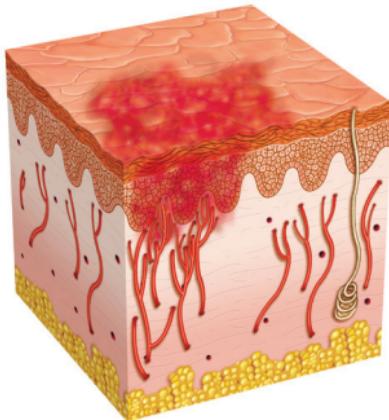
- A transient, elevated, irregularly shaped, localized lesion of the skin due to dermal edema; most wheals are red, pale pink, or white.

9. Petechia

- A <5 mm diameter macule resulting from a deposition of blood into the skin. The term purpura is sometimes used for such lesions that are somewhat larger, which may also be palpable.

10. Ecchymosis

- A larger area of discolored skin resulting from bleeding into the skin.



11. Telangiectasis

- Visibly dilated, superficial, cutaneous blood vessels.

12. Comedo (black or whitehead)

- A white, gray, or black noninflammatory plug in the follicle.

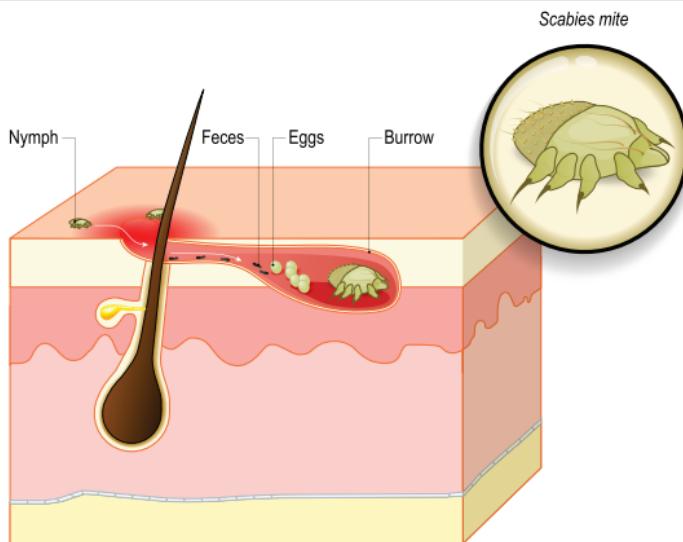


BLACKHEAD

WHITEHEAD

13. Burrow

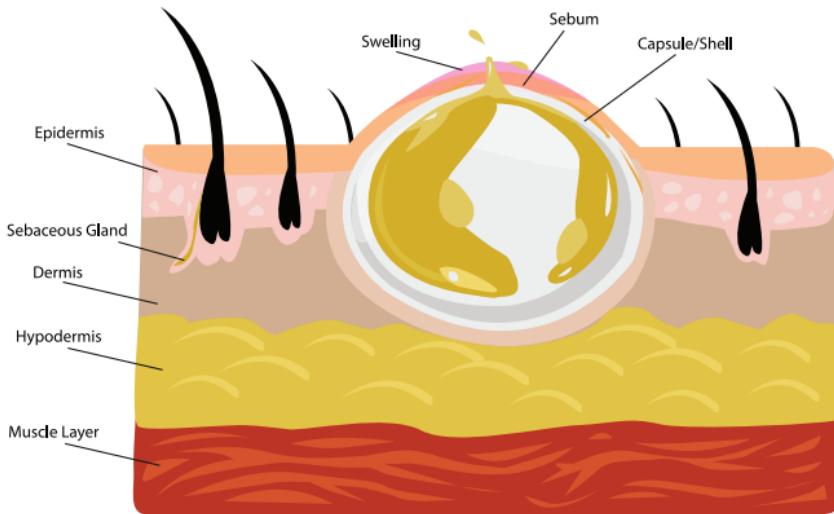
- A tunnel, tract, or passage in skin made by parasites as the mite of scabies and the larvae of larva migrans.



Scabies mite

14. Cyst

- A non-inflammatory collection of fluid or semisolid material surrounded by a well-defined wall.



■ B. SECONDARY LESIONS

1. Scale

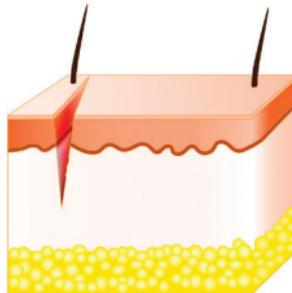
- A visible accumulation of keratin, forming a flat plate or flake

2. Crust (scab)

- Dried serum, blood or pus on the surface of the skin.

3. Fissure

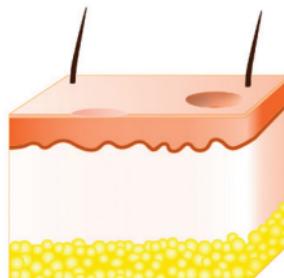
- A crack in the skin.
-



Fissure

4. Erosion

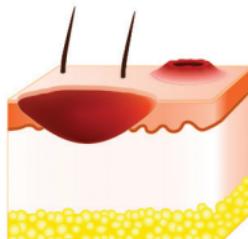
- Loss of either a portion of or the entire epidermis.
-



Erosion

5. Ulcer

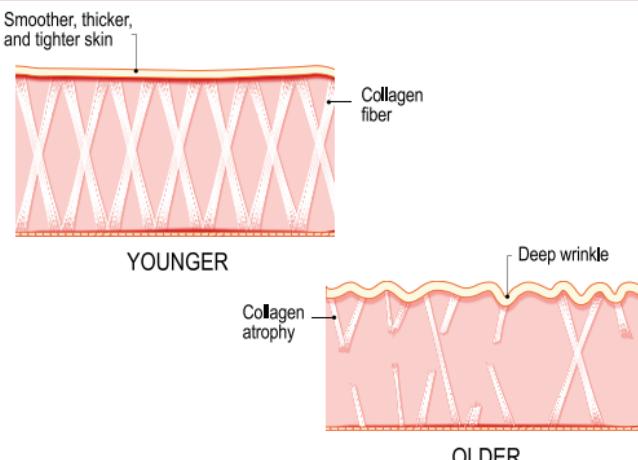
- Full-thickness loss of the epidermis plus at least a portion of dermis; may extend into the subcutaneous tissue.



Ulcer

6. Atrophy

- A disappearance, or "wasting," of tissues or parts of tissues.



7. Excoriation (*scratch mark*)

- A loss of the epidermis and a portion of the dermis due to scratching or an exogenous injury.

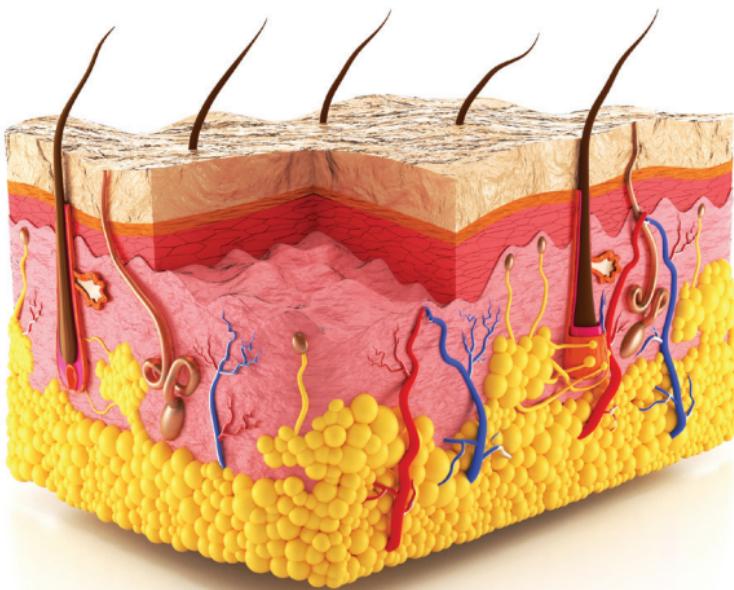
8. Scar

- A fibrotic residual of a previous inflammatory process.



SECTION 3

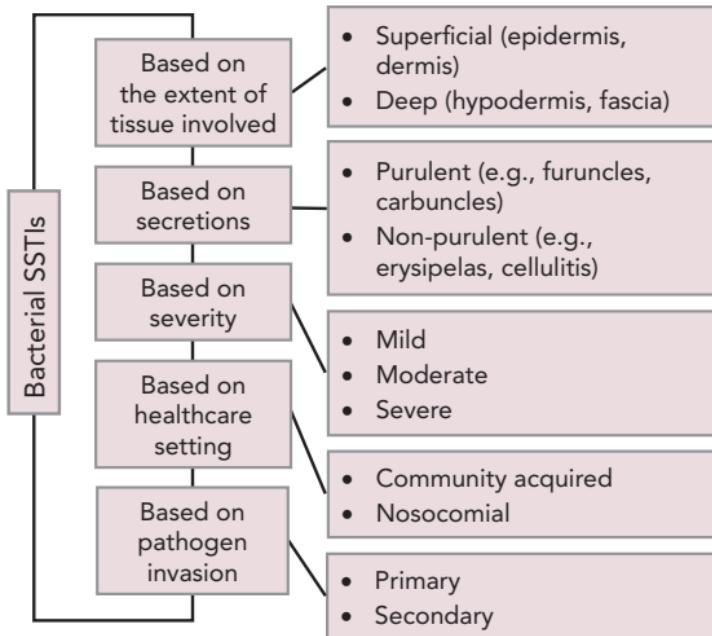
BACTERIAL INFECTIONS



■ BACTERIAL SKIN AND SOFT TISSUE INFECTIONS (SSTIs)

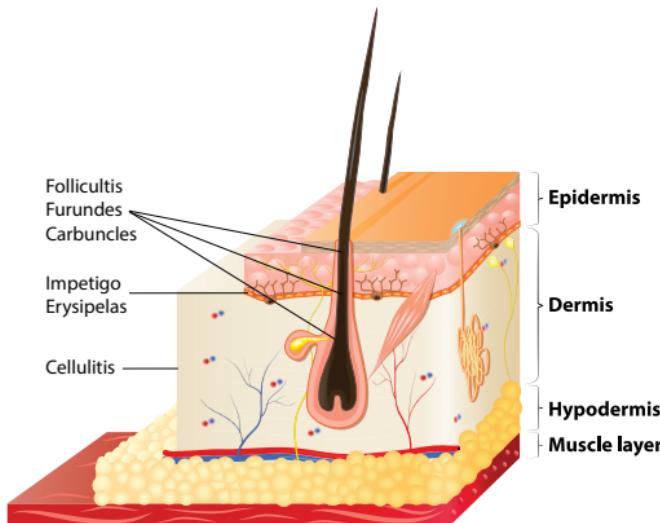
Bacterial skin and soft tissue infections (SSTIs) can have a wide range of presentations, and can be classified in a number of ways. Nevertheless, traditionally, they are divided into uncomplicated and complicated SSTIs (based on presence of certain conditions like diabetes mellitus or other immunosuppressed states). Accordingly, uncomplicated SSTIs such as folliculitis or carbuncles would be considered complicated if patients have underlying comorbidities impacting the management.

Classification of bacterial SSTIs



Another classification typically impacting the management of bacterial SSTIs is based on the manner of infection development, and thus classifying the infections as either ‘primary’ or ‘secondary’ bacterial infections. It suggests that primary SSTIs occur when microorganisms invade otherwise healthy skin, whereas secondary SSTIs occur when, because of underlying disease, microorganisms infect already damaged skin.

Human skin structures and corresponding locations of SSTIs



■ COMMON PRIMARY BACTERIAL SKIN INFECTIONS

The four most common primary bacterial skin infections are impetigo, erysipelas, cellulitis, and folliculitis.

Common primary bacterial skin infections

Impetigo

Erysipelas

Cellulitis

Folliculitis

■ IMPETIGO

Impetigo is a common localized acute bacterial skin infection, characterized by multiple erythematous, vesicular, and pruritic lesions. It presents as a superficial blister atop a red macule, which quickly evolves to form eroded, exudative, yellow “honey-crusted” lesions. Clinically, infection in impetigo becomes apparent approximately 10 days after colonization with bacteria. The condition mainly affects exposed areas of the body, such as the face and extremities, but may also affect trunk, perineum and other body sites.

There are two main forms of impetigo, recognized on the basis of clinical, bacteriological, and histological findings:

- Non-bullous or crusted impetigo (distinct yellow, crusting lesions), and
- Bullous impetigo (bullae that rupture to form a brown crust).

Non-bullous impetigo

The non-bullous impetigo is the most common form, most often occurs in young children (aged 2 to 5 years), on the face or extremities. It presents with small fluid-filled vesicles that soon develop into pustules that rupture, leaving golden-yellow crusts. The condition is caused by *S. pyogenes* alone or as part of a mixed infection with *S. aureus*. Untreated impetigo usually resolves within 2 to 4 weeks without scarring. However, treatment may be justified to speed-up healing of the lesions, decrease their spread, and shorten the duration of contagious period.

Non-bullous impetigo



Bullous impetigo

Bullous impetigo is associated with *S. aureus*, and is characterized by superficial, thin-walled, and bullous lesions. It presents with vesicles that develop into yellow fluid-filled bullae that rupture, leaving brown

crusts. The thin, transparent, varnish-like crust that appears on rupturing of the lesions can be distinguished from the stuck-on crust of common impetigo. This distinctive appearance of bullous impetigo results from local action of the epidermolytic toxin (exfoliation) - it is considered that bullous impetigo is due to staphylococcal exfoliative toxins (exfoliatin A–D), which target desmoglein 1 (a desmosomal adhesion glycoprotein), and cleave off the superficial epidermis through the granular layer. No trauma is required, as the bacteria can infect intact skin.

Bullous Impetigo (Severe)



Deeper impetigo (ecthyma)

A third, deeper, form of impetigo is known as ecthyma, in which lesions (ulcerations) form beneath the crusted plaques. The lesions usually occur on the legs and other covered areas of the body; often occurring as a complication of debility and infestation. Ecthyma starts as non-bullous impetigo but develops into a punched-out necrotic ulcer; ulcers have a punched-out appearance when the crust or purulent materials are removed. It is usually caused by *S. pyogenes*, but co-infection with *S. aureus* may occur. Lesions in ecthyma generally heal slowly and leave scars.

Ecthyma wound



■ HAIR FOLLICLE INFECTIONS

Folliculitis, furuncles and carbuncles

Folliculitis is an infection of one or more hair follicles that may affect any area of the body with hairs; thus excluding the palms and soles.

In **folliculitis**, a small red bump, or pimple, develops at the site of the involved hair follicle, and this may be associated with rash or pruritus. Folliculitis can be further divided into two major categories on the basis of histological location: superficial and deep.

The presentation thus depends on its severity, which ranges from superficial inflammation of an individual hair follicle to a deeper infection of the follicle (**furuncle**) to clusters of coalescing abscesses found deeper in the subcutaneous tissues (**carbuncles**). Common bacterial causes of folliculitis include *S. aureus*, *S. pyogenes*, *Pseudomonas* species, and *Proteus* species.

Folliculitis



SECTION 3

Furuncle



Carbuncle



The most superficial form of skin infection is staphylococcal folliculitis, caused by *S. aureus*, and manifested by tiny erythematous follicular pustules without involving the surrounding skin. It usually presents at scalp and extremities as red, often itchy, papules and/or pustules at the base of the hair shaft.

In deep folliculitis, infection extends deeply into the follicle, and the resulting peri-folliculitis causes a more marked inflammatory response than that seen in superficial folliculitis.

Furuncles and carbuncles occur as the follicular infection progresses deeper and extends out from the follicle. Commonly known as an abscess or boil, a furuncle represents a tender, erythematous, firm or fluctuant mass of walled-off purulent material, arising from the hair follicle.

These lesions may occur anywhere on the body, but have a predilection for hairy areas that are exposed to friction and macerations. Pus may drain from the boil along with a plug of inflammatory cells and dead tissue. The pathogen is usually *S. aureus*.

Furuncles may progress to carbuncles, which is an aggregate of furuncles penetrating to deeper layers of skin, forming broad, swollen, erythematous, deep, and painful masses that usually open and drain through multiple tracts.

A carbuncle thus typically represents a confluence of boils, as a large erythematous, indurated painful lesion with multiple draining sites; these pus draining openings are often associated with fever, swollen lymph nodes, and fatigue. Carbuncles usually develop in areas of the body where the skin is thick, such as the back of the neck.

Cellulitis

Cellulitis is a painful, erythematous infection of the dermis and subcutaneous tissues that is characterized by warmth, edema, and advancing borders. It commonly occurs near breaks in the skin, such as trauma, tinea infections, or ulcerations, but may occasionally present in the normal appearing skin; the pathogen generally invades through a breach in the skin surface, and infection is promoted by presence of tissue edema. *Cellulitis* is usually caused by *S. pyogenes* or *Staphylococcus* species.

Cellulitis



Common physical findings in cellulitis include erythema, edema, warmth, and tenderness of the affected area. In addition, patients may also experience fever, tender lymphadenopathy, and abscess formation, especially if *S. aureus* is implicated as the causative agent. Unlike erysipelas, the involved area in cellulitis is poorly demarcated. Cellulitis is generally considered a serious infection because of the propensity

of the microorganism(s) to invade lymphatic tissue and blood; if left untreated, it can progress to adjacent tissues and cause an abscess, septic arthritis, or osteomyelitis.

Erysipelas

Erysipelas, also known as St. Anthony's fire, is a superficial form of cellulitis with sharply demarcated borders. It usually presents acutely as an intensely erythematous infection, often with associated lymphatic streaking (involving dermis and dermal lymphatics). Erysipelas is often confused with cellulitis; however, there are few features, which may help in differentiating the two. Clinically, it is more superficial, and is characterized by a bright red erythematous lesion with a sharp margin as opposed to the undefined border of cellulitis.

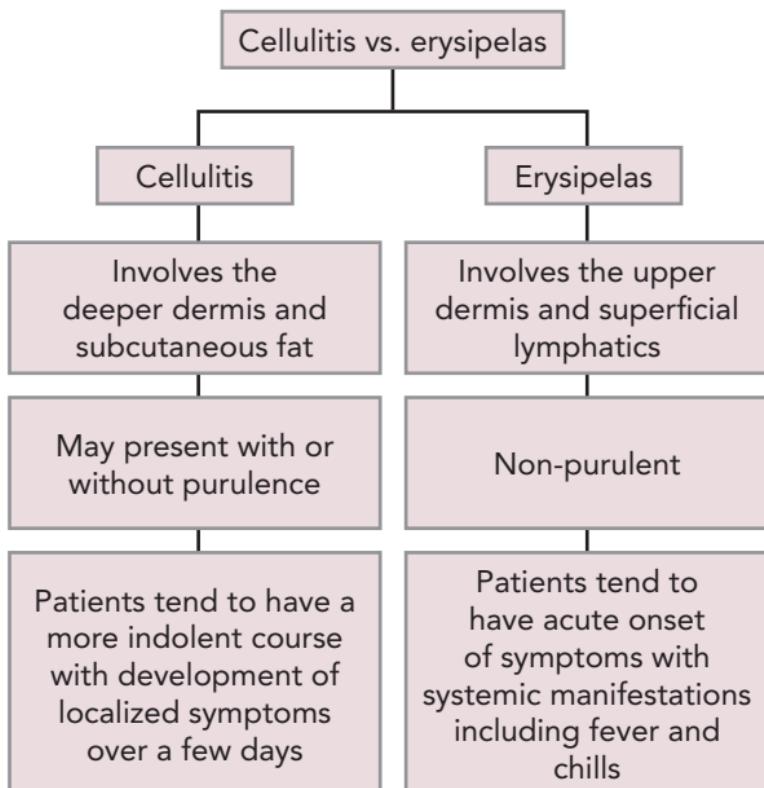
Erysipelas



SECTION 3

This condition is often associated with fever, burning pain, and lymphangitis, caused by prominent lymphatic compromise of the affected area. However, lymphatic spread and subsequent bacteremia are rarely found because of the superficial nature of the disease, making blood culture collection unnecessary in suspected cases. The most common pathogen causing erysipelas is *S. pyogenes*.

Differentiating features of cellulitis and erysipelas



Contrary to the earlier trends which suggest facial involvement as the most common presentation, erysipelas often develops on the lower extremities. Predisposing factors for this condition include *S. pyogenes* colonization of the skin or recent oropharyngeal infection, dermatophyte infection between the toes or of the toenails (i.e., tinea pedis), chronic venous stasis, and preexisting leg ulcers.

■ DIAGNOSIS OF BACTERIAL SSTIs

Usually, as in most cases the diagnosis of SSTIs is based on clinical impression, the first step is the clinical suspicion of an SSTI; laboratory investigations then may help to confirm the diagnosis and elucidate characteristics of specific etiologies, but are seldom required as they are unlikely to change the management of localized SSTIs in otherwise healthy patients.

The minimum criterion here is a skin lesion with cardinal signs of an SSTI, such as erythema, edema, tenderness to palpation, and increased warmth; while in some cases the affected area may also become dysfunctional depending on the severity of infection. The symptom that highly increases the suspicion of an SSTI is fever, and it is more likely to be present in deeper infections.

Other signs and symptoms, such as fluctuance, crepitus, induration, blisters, or bullae may help the clinician determine the depth of infection or the presence of an abscess, thus helping to augment the clinical suspicion and confirm the diagnosis. Overall, in most cases of bacterial SSTIs, clinical examination and laboratory investigations (staining and/or culturing of a specimen of pus or exudates) is often adequate for making the diagnosis.

■ TREATMENT OF BACTERIAL SSTIs

Most bacterial SSTIs common in primary care can often be managed in an outpatient setting; and only in few cases more urgent care or inpatient management would be required. The treatment requires antibacterial therapy that may be administered either topically or systemically, depending on the extent and severity of the infection.

In this context, data supports the use of topical antibiotics (generally given for 5 days) for simple infections confined to skin and underlying superficial soft tissues, such as non-complicated impetigo; whereas systemic antibiotics covering Gram-positive cocci are recommended for complicated cases of impetigo and deeper non-purulent SSTIs.

For purulent infections, incision and drainage (I & D) is a key management component. Furthermore, it is important to include basic wound care techniques, including dressings, as part of overall management of SSTIs.

Some common topical antibacterial agents for SSTIs	
Agent	Dosing regimen
Fusidic acid	Localized pyoderma: Apply topical fusidic acid 2% cream up to 3 times daily for up to 10 days
Mupirocin	Skin infections ≥ 2 months: Apply to affected area twice daily for 5 days MRSA decolonization ≥ 12 years: Apply to anterior nares twice daily for 5 days [MRSA: methicillin-resistant S. aureus]
Retapamulin	Impetigo ≥ 9 months: Apply to affected area twice daily for 5 days

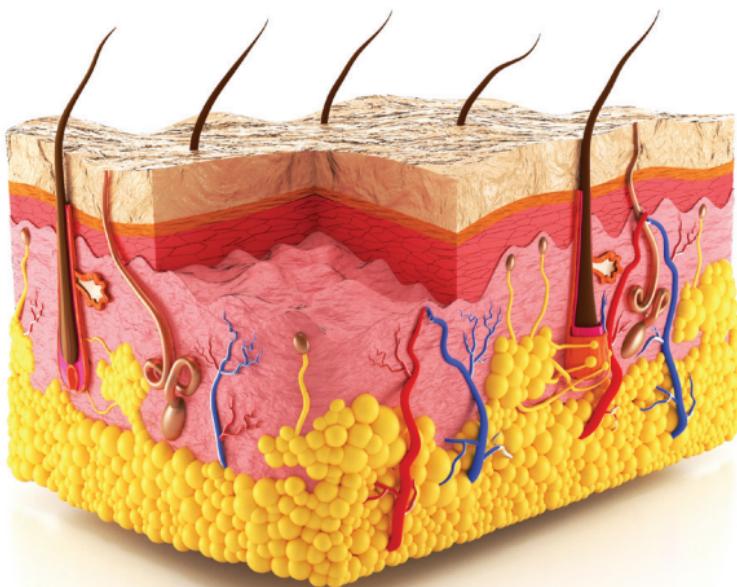
Some common systemic antibacterial agents for SSTIs	
Agent	Dosing regimen
Amoxicillin/ clavulanic acid	Adults and children ≥ 40 kg: 875 mg of amoxicillin PO twice daily Children < 40 kg: 25 mg/kg/day of amoxicillin PO divided into 2 doses
Cefazolin	Adults: 1 g IV three times daily Children: 50 mg/kg/day IV divided into 3 doses
Ceftriaxone	Adults: 1 g IV daily Children: 50–75 mg/kg/day IV divided into 1 to 2 doses
Clindamycin	Adults: 300–450 mg PO four times daily Adults: 600 mg IV three times daily Children: 20–40 mg/kg/day IV/PO divided into 3 doses
Dicloxacillin	Adults and children ≥ 40 kg: 250–500 mg PO four times daily Children < 40 kg: 25–50 mg/kg/day PO divided into 4 doses
Doxycyline (Not active against <i>S. pyogenes</i>)	Adults and children > 45 kg: 100 mg PO twice daily Children ≥ 8 years and ≤ 45 kg: 2 mg/kg PO twice daily
Linezolid	Adults and children ≥ 12 years: 600 mg IV/PO twice daily Children < 12 years: 10 mg/kg/day IV/PO twice daily

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Some common systemic antibacterial agents for SSTIs	
Minocycline (Not active against <i>S. pyogenes</i>)	Adults: 200 mg PO on day 1 followed by 100 mg twice daily Children \geq 8 years: 4 mg/kg PO on day 1 followed by 2 mg/kg twice daily
Penicillin G	Adults: 2–4 million units IV four to six times daily Children: 60,000 to 100,000 units/kg/dose IV four times daily
Penicillin VK	Adults: 250–500 mg PO four times daily Children: 25–50 mg/kg/day PO divided into 2 to 4 doses
Piperacillin-tazobactam	Adults and children $>$ 40 kg: 3.375 g IV three or four times daily Children \leq 40 kg: 100 mg/kg of piperacillin IV three times daily
Trimethoprim/ Sulfamethoxazole (Not active against <i>S. pyogenes</i>)	Adults: 1 to 2 DS tablet(s) PO twice daily [DS: double strength (160 mg of trimethoprim and 800 mg of sulfamethoxazole)] Children: 8–12 mg/kg/day of trimethoprim divided into 4 doses IV or 2 doses PO
Vancomycin	Adults: 30 mg/kg/day divided into 2 doses Children: 40 mg/kg/day divided into 4 doses

SECTION 4

FUNGAL & YEAST INFECTIONS



■ INTRODUCTION TO FUNGAL DISEASES

Fungal diseases or infections affect a large proportion of the population, and are a significant cause of morbidity and mortality. The spectrum of fungal infections is extensive, resulting in a wide variety of diseases of varying severity; dependent on the pathogen virulence and the hosts' immunity status.

Some medically important fungi (Molds)

Group	Disease	Etiological agent
Molds Black fungi	Chromoblastomycosis	<i>Cladosporium carriionii</i> <i>Fonsecaea pedrosoi</i>
	Phaeohyphomycosis	<i>Exophiala jeanselmei</i> <i>Wangiella dermatitidis</i> <i>Xylohypha bantiana</i>

Some medically important fungi (Dermatophytes)

Group	Disease	Etiological agent
Dermatophytes	Tinea capitis	<i>Microsporum canis</i> <i>Trichophyton tonsurans</i>
	Tinea corporis	<i>Microsporum gypseum</i> <i>Trichophyton mentagrophytes</i> <i>Trichophyton rubrum</i>
	Tinea cruris	<i>Epidermophyton floccosum</i>
	Tinea pedis	<i>Trichophyton mentagrophytes</i> <i>Trichophyton rubrum</i>

Some medically important fungi (Dimorphic)		
Group	Disease	Etiological agent
Dimorphic	Blastomycosis	<i>Blastomyces dermatitidis</i>
	Coccidioidomycosis	<i>Coccidioides immitis</i>
	Histoplasmosis	<i>Histoplasma capsulatum</i>
	Paracoccidioidomycosis	<i>Paracoccidioides brasiliensis</i>
	Sporotrichosis	<i>Sporothrix schenckii</i>

Some medically important fungi (Yeasts)		
Group	Disease	Etiological agent
Yeasts	Candidiasis	<i>Candida albicans</i> <i>Candida tropicalis</i>
	Cryptococcosis	<i>Cryptococcus neoformans</i>
	Pityriasis versicolor	<i>Malassezia furfur</i>

■ SPECTRUM OF FUNGAL INFECTIONS: CLASSIFICATION AND COMMON PRESENTATIONS

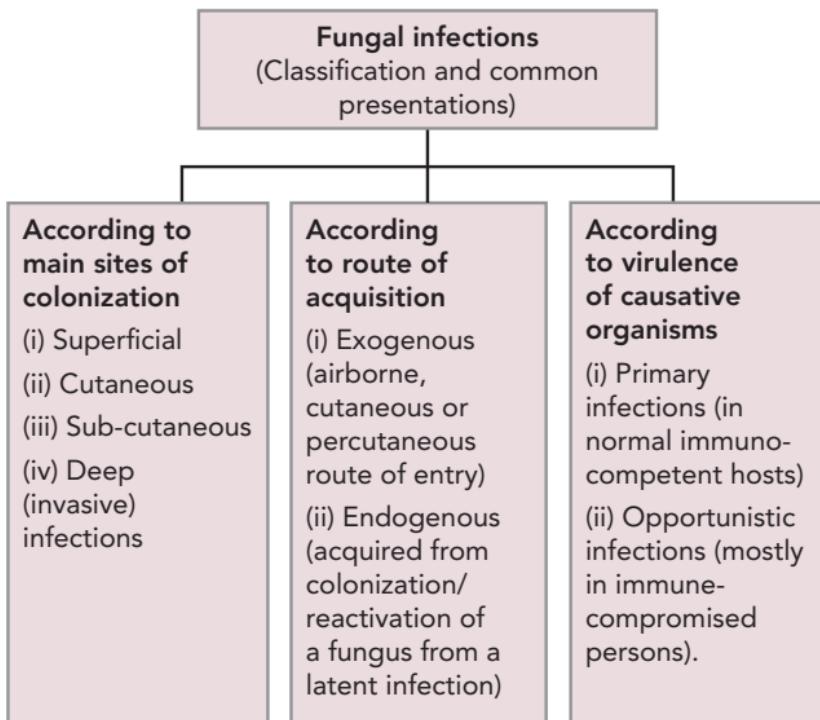
There are different clinical nomenclatures used to describe fungal infections, essentially based on the following criteria:

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- I Site of the infection;
- II Route of acquisition of the pathogen; or
- III The type of virulence exhibited by the fungus.

Usually, most pathogenic fungi are classified by tissue levels that are the main sites of colonization. These infections can thus present clinically in following general manners - ***superficial, cutaneous, subcutaneous, or deep*** (invasive) infections, dependent on the type and degree of tissue involvement and hosts' response to pathogen.

Spectrum of fungal infections



Superficial infections

Superficial infections are categorized such given the fact that they are limited to the stratum corneum layer. Broadly, following fungal infections and their main etiological agents are included in this category:

- Black piedra (*Piedraia hortae*),
- White piedra [*Trichosporon beigelii* (*T. asahii*)],
- Pityriasis versicolor or Tinea versicolor (*Malassezia furfur*), and
- Tinea nigra [*Hortaea werneckii* (*Phaeoannellomyces werneckii*)].

Pityriasis versicolor/Tinea versicolor



Cutaneous (Dermatophyte) infections

Cutaneous fungal infections are caused by fungi that colonize the skin, hair, and nails on the living host (dermatophytes) and possess greater invasive properties than those causing superficial infections, yet they

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are keratinophilic and limited to the keratinized tissues; besides, there is tissue inflammation, which can be elicited by the organism or its products. Broadly, cutaneous fungal infections may be classified as:

- i. *Dermatophytoses*, which are caused by agents of the genera *Epidermophyton*, *Microsporum*, and *Trichophyton*, or as
- ii. *Dermatomycoses*, which are cutaneous infections attributable to other fungi, the most common of which are *Candida* spp.

In humans, *Trichophyton rubrum* is the main causative agent of Dermatophytoses group of diseases. Infections caused by dermatophytes are called “tineas” and are classified according to the affected site.

Main types of “tinea” infections		
Tinea	Main Dermatophyte	Site of infection
Tinea capitis	<i>T. tonsurans</i> <i>Microsporum canis</i> <i>Trichophyton violaceum</i> <i>Trichophyton soudanense</i>	Scalp
Tinea corporis	<i>Trichophyton rubrum</i> <i>T. tonsurans</i> <i>Microsporum canis</i>	Body (chest, face, arms, and/or legs)
Tinea cruris	<i>T. rubrum</i>	Groin folds
Tinea pedis (athlete's foot)	<i>T. rubrum</i> <i>T. interdigitale</i> <i>Epidermophyton floccosum</i>	Foot (soles or interdigital spaces)
Tinea unguium (onychomycosis)	<i>T. rubrum</i> <i>T. interdigitale</i>	Nails

Subcutaneous infections

There are three general types of subcutaneous fungal infections - chromoblastomycosis, mycetoma, and sporotrichosis; all of which appear to be caused by traumatic inoculation of the etiological fungi into the subcutaneous tissue. Although most of the fungi implicated in this category exist in a hyphal morphology, the agents causing chromoblastomycosis and sporotrichosis are exceptions.

Chromoblastomycosis & mycetoma

Chromoblastomycosis is characterized by skin lesions, which can present clinically in five different forms: nodular, tumoral lesions, verrucous, plaque and cicatricial; on histological examination, characteristic dark-colored, thick-walled, muriform cells i.e., sclerotic cells (Medlar bodies) are observed, which is a histopathological criterion for the diagnosis. In contrast, mycetoma represents a progressive subcutaneous granulomatous infection, which is destructive to the adjacent subcutaneous tissue, muscle, and bone. Mycetoma is characterized by presence of draining sinus tracts from which small but grossly visible pigmented grains are extruded, which are essentially microcolonies of the fungi causing the infection.

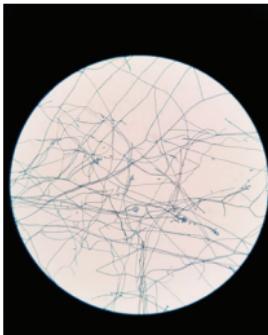
Chromoblastomycosis usually results from a traumatic injury and inoculation of microorganism from specific group of dematiaceous fungi (usually *Fonsecaea pedrosoi*, *Phialophora verrucosa*, *Cladophialophora carrionii*). In contrast, mycetoma has more diverse causes, and can be classified as eumycotic and actinomycotic mycetoma. Both eumycetoma and actinomycetoma present as a progressive, subcutaneous swelling, although actinomycetoma has a more rapid course. Common agent for eumycotic mycetoma is *Pseudallescheria boydii*, and that of actinomycotic

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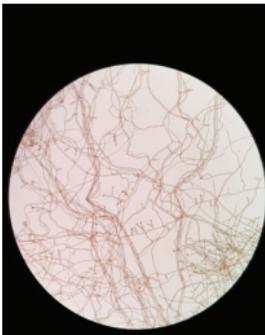
mycetoma is *Nocardia brasiliensis*. Many of the fungi causing mycetoma are dematiaceous (melanized; pigmented brown to black) fungi.

Fungi causing Chromoblastomycosis

Fonsecaea pedrosoi



Phialophora verrucosa



Cladophialophora carrionii



Sporotrichosis

Sporotrichosis - a chronic granulomatous mycotic infection - is the next class of subcutaneous fungal infections, which occurs due to the dimorphic *Sporothrix schenckii*, generally by traumatic inoculation of soil, plants, and organic matter contaminated with the fungus. The infection usually spreads along cutaneous lymphatic channels of the extremity involved. In many cases, after a variable incubation period, progressively enlarging papulo-nodule at the inoculation site develops that may ulcerate (fixed cutaneous sporotrichosis), or multiple nodules appear proximally along lymphatics (lymphocutaneous sporotrichosis).

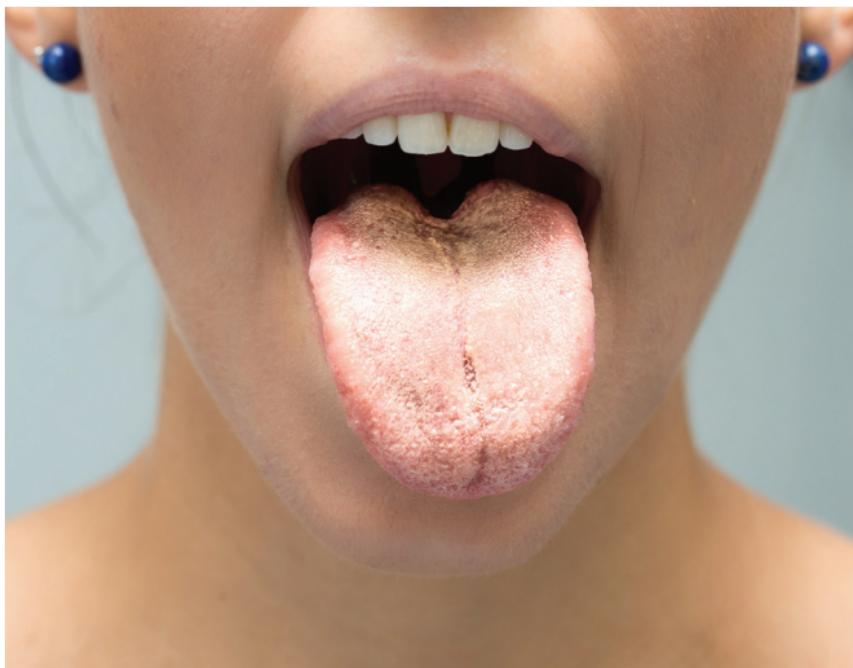
Sporotrichosis



Candidiasis

Candidiasis is the most common opportunistic fungal infection, and can be caused by several *Candida* species, including *Candida albicans*, *C. glabrata*, *C. parapsilosis*, *C. tropicalis*, and *C. krusei*. The infection may occur as superficial or deep, with the former involving epidermal and mucosal surfaces.

Candidiasis (*Candida albicans*) infection on tongue



■ DIAGNOSIS OF FUNGAL INFECTIONS

The prompt diagnosis of fungal infections often remains a challenge given the fact that they often present with non-specific symptoms and signs. Besides, simple colonization is often difficult to distinguish from active infections. Therefore, confirmatory diagnosis of fungal infections often depends on more informative laboratory techniques, which include both direct methods like microscopy and histological examination, and indirect methods like Wood's lamp examination. In most cases, however, clinical presentation is often the first criteria that raise suspicion of a fungal infection and direct further investigations.

Herein, the success of a laboratory confirmation of clinically diagnosed fungal infection frequently relies on proper collection of specimens for microscopic examination and culture. Direct microscopic examination of a potassium hydroxide (KOH) mounted preparation is the most simple and important test for diagnosing superficial fungal infection and dematiaceous fungal infection.

■ TREATMENT OF FUNGAL INFECTIONS

Control of fungal infections includes both prevention and treatment. Prevention mainly encompasses avoidance of risk factors such as environments and conditions conducive to fungal growth. Clinical outcomes for individual patients with fungal infections are however often better when appropriate (antifungal) treatment is initiated on time. Nevertheless, the management could be dependent on not only the specific pathogen but also the host disease status. Herein, use of currently available main antifungals like azoles in combination with other antifungals with distinct mechanisms of action is likely to provide enhanced efficacy in treatment of wide array of fungal infections.

Pharmacological treatments for superficial fungal infections can be grouped into topical and systemic. Generally, common infections like tinea pedis, tinea corporis, and tinea cruris respond well to topical therapy. Treatment course for topical treatment spans from as short as 1 week to 4-6 weeks. Oral therapy can be used for these infections if the infection is extensive, severe, or recalcitrant. Tinea capitis must however be treated with oral antifungal therapy, since topical agents do not penetrate the hair shaft. Tinea unguium responds better to oral therapy than to topical treatment.

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Topical agents for fungal skin infections

Azoles	<ul style="list-style-type: none">• Clotrimazole• Econazole• Efinaconazole• Ketoconazole• Luliconazole• Miconazole• Oxiconazole• Sertaconazole• Sulconazole
Allylamines	<ul style="list-style-type: none">• Naftifine• Terbinafine hydrochloride
Benzylamine	<ul style="list-style-type: none">• Butenafine hydrochloride
Other	<ul style="list-style-type: none">• Ciclopirox• Tolnaftate

Systemic treatment options for Pityriasis versicolor

- Ketoconazole 200mg/day for 10 days
- Fluconazole 150mg/week for 3 weeks
- Itraconazole 200mg/day for 7 days

Systemic treatment of dermatophyte infections	
Tinea pedis (dry type)	<p>First line Terbinafine 250 mg/day for 2 weeks Itraconazole 200–400 mg/day for 1 week</p> <p>Alternative Fluconazole 6 mg/kg/week for 4–6 weeks</p>
Tinea corporis (extensive)	<p>First line Terbinafine 250 mg/day for 1 week Itraconazole 200 mg/day for 1 week</p> <p>Alternative Fluconazole 150–200 mg/week for 2–4 weeks</p>
Onychomycosis due to dermatophytes*	<p>First line Terbinafine 250 mg/day for 12 weeks (toe nails) or 6 weeks (fingernails) Itraconazole 200 mg bid for 1 week/month for 3 months (toe nails) or 2 months (finger nails)</p> <p>Alternative Fluconazole 150–200 mg/week for 6–9 months (toe nails) or 3–4 months (finger nails)</p>

Systemic treatment of dermatophyte infections

Tinea capitis
(children)

First line

Terbinafine 125 mg (<25 kg), 187.5 mg (25–35 kg) or 250 mg (>35 kg) daily for 3–4 weeks

Mainly *Trichophyton* infections

Griseofulvin 10–15 mg/kg/day for 6–8 weeks

Mainly *Microsporum* infections

Alternative

Itraconazole 5 mg/kg/day (maximum 500 mg) for 4–8 weeks

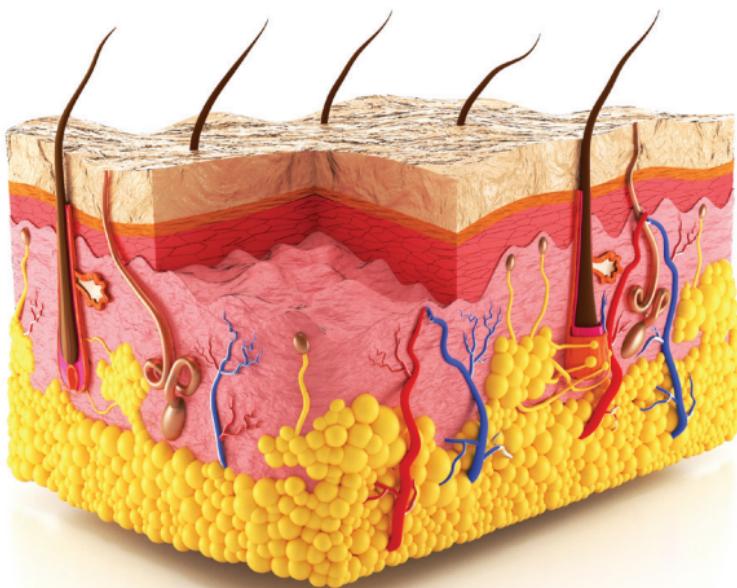
* In extensive infections, including those involving the nail matrix, combination with an oral and topical antifungal is useful.

Systemic treatment options for oral/vulvovaginal candidiasis

- Itraconazole 200mg/day for 5 days
- Fluconazole 150mg single dose
- Ketoconazole 200mg/day for 5-10 days

SECTION 5

MIXED SKIN INFECTIONS



■ MIXED SKIN INFECTIONS

Skin and skin-structure infections are common in ambulatory settings, with many of them frequently mixed in nature, characterized by presence of more than one type of infection and/or lesions. Such acute skin infections can often involve a mix of several different pathogens, and are a common reason for seeking care. Indeed, they have become significant causes of acute skin infections.

These mixed infections are not limited to a mix of gram-positive and gram-negative bacterial pathogens or mixed mycotic infections, but can also encompass a range of presentations emerging from a concurrence of bacterial and fungal co-infections.

■ BACTERIAL AND FUNGAL INTERACTIONS

Humans are colonized by varied populations of bacterial and fungal pathogens in both a healthy state and in disease settings, and interactions between them can often be detrimental to the host. Indeed, co-infections arising from a concurrence of bacteria and fungi have been implicated in enhanced host colonization and virulence.

Besides providing attachment sites for different species, bacterial-fungal communities can create environmental conditions that promote or control the growth of other microbes. For instance, a considerable proportion of such microbial infections might be biofilm-associated, wherein formation of mixed-species biofilms could create a protected environment, facilitating different bacterial-fungal interactions while also allowing for survival to external assaults. As follows, these infections can correlate with increased frequency or severity of the disease. Furthermore, in many such cases, it is not always clear which organism is responsible for the initial infection and which organism represents a secondary infection.

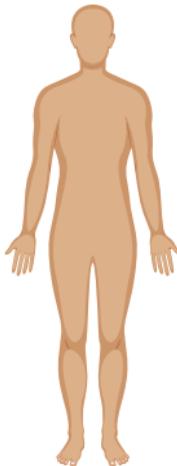
Some important sites for bacterial–fungal interactions (including Skin)

Burn wound site

- Gram-negative bacteria (usually *Pseudomonas* spp.)
- Gram-positive bacteria (usually *Staphylococcus* spp.)
- Fungi (usually *Candida* spp. but sometimes *Aspergillus* spp.)

Skin

- Gram-positive and Gram-negative bacteria (usually *Staphylococcus* spp.)
- *Candida* spp.



Intra-abdominal site

- Gram-negative bacteria (usually Enterobacteriaceae)
- Gram-positive bacteria (usually *Enterococcus* spp.)
- *Candida* spp.

Oral cavity

- Gram-positive bacteria (usually *Streptococcus* spp.)
- *Candida* spp.

Lower reproductive tract

- Gram-negative bacteria
- Gram-positive bacteria
- Fungi (usually *Candida* spp. but sometimes *Cryptococcus neoformans*)

Urinary site with catheters

- Gram-negative bacteria (*Pseudomonas* spp. and Enterobacteriaceae)
- Gram-positive bacteria
- *Candida* spp.

■ MANAGEMENT OF MIXED BACTERIAL AND FUNGAL INFECTIONS

Identifying mixed bacterial-fungal infections - through methods appropriate as described for respective pathogens - means that a combination therapy comprising both antibacterial and antifungal antibiotics may be required. For instance, in intertrigo, a superficial inflammatory dermatitis/rash that usually affects the folds of the skin and frequently creates an entry point for secondary fungal and bacterial infections, fungal lesions can be treated with topical nystatin, clotrimazole, ketoconazole, oxiconazole, or econazole, and secondary streptococcal infections can be treated with topical mupirocin or oral penicillin.

Treatment options for inflammatory and infectious intertrigo	
Condition	Treatments
Intertrigo	Topical: zinc oxide ointment, petrolatum, talcum powder, aluminum sulfate
Intertrigo complicated by secondary bacterial infections (Erythrasma)	Topical: erythromycin, clindamycin, Whitfield ointment, chlorhexidine Oral: erythromycin
Intertrigo complicated by secondary bacterial infections (Group A beta-hemolytic streptococcus)	Topical: mupirocin, erythromycin, low-potency steroids Oral: penicillin, cephalexin, ceftriaxone, cefazolin, clindamycin

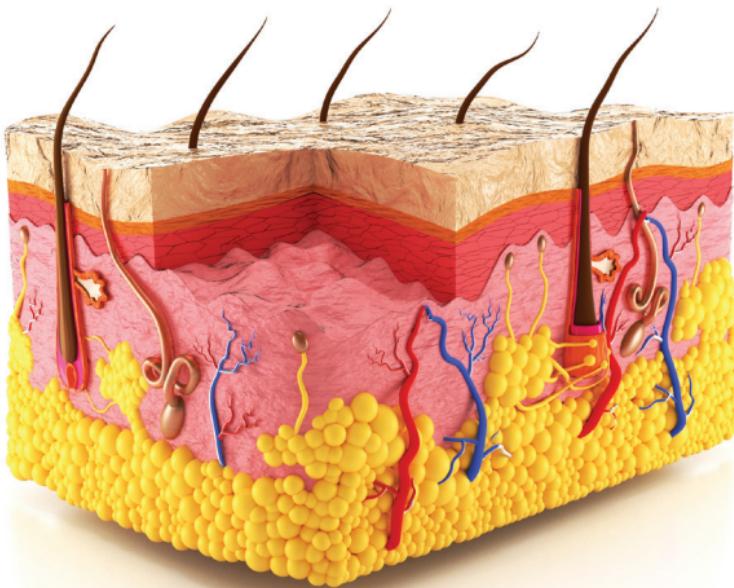
Treatment options for inflammatory and infectious intertrigo	
Intertrigo complicated by secondary fungal infections (Candida)	Topical: nystatin, clotrimazole, ketoconazole, oxiconazole, econazole Oral: fluconazole
Intertrigo complicated by secondary fungal infections (Dermatophytes)	Topical: clotrimazole, ketoconazole, oxiconazole, econazole

Combination preparations of topical antibiotics + corticosteroids + antifungal may provide a convenient option for patients with eczema complicated by *S. aureus*/candida infections. Triple combination of antifungal, antibacterial and potent steroid has also been found to be efficacious and tolerable in reducing signs and symptoms (scaling, inflammation, burning and itching) of eczematous disorder associated with underlying tinea/yeast infection.

Herein, products with biopolymer may be more effective than products without biopolymer in achieving clinical improvement or resolution of skin infection conditions. This could possibly be attributable to the better pharmacokinetics of the biopolymers that gives them an advantage over the conventional preparations. Such products with biopolymer are likely to have a better safety and efficacy profile than those without biopolymer, and may help to achieve better compliance with the treatment.

SECTION 6

VIRAL INFECTIONS



■ VIRAL SKIN DISEASES

Viral diseases of the skin present a unique challenge to the skin's immune system, and can result in both localized and generalized skin infections. These infections can be caused by viruses from several major groups.

Generally, viral skin diseases can be classified according to their clinical features into following three types:

1. Degeneration of epidermal cells and blistering (e.g., in herpes simplex and herpes zoster),
2. Tumorous changes in epidermal cells (e.g., in verruca vulgaris), and
3. Allergic eruptions on the whole body (e.g., in measles and rubella).

While the first two types are caused by a viral infection in epidermal keratinocytes; the last type is caused by a systemic viral infection (viremia). Amongst the various viral skin diseases affecting humans, herpes simplex virus (HSV) infection is probably the most common.

Viral skin diseases and associated viruses	
Disease	Virus
Localized disease	
Herpes labialis and herpes genitalis	Herpes simplex virus
Herpes zoster	Varicella zoster virus
Vaccinia	Vaccinia virus
Molluscum contagiosum	Molluscum contagiosum virus
Warts	Papillomavirus

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Viral skin diseases and associated viruses	
Generalized disease	
Measles	Measles virus
Rubella	Rubella virus
Enteroviral exanthema and enanths	Several enteroviruses
Erythema infectiosum	Parvovirus

Skin viruses, their cell tropism, and the populations vulnerable to infection		
Virus	Cell tropism	Vulnerable populations
Herpes Simplex Virus	Epidermal keratinocytes	<ul style="list-style-type: none">• Neonates• Immunocompromised• Males – HSV-1• Females – HSV-2
Vaccinia Virus	Dendritic cells, macrophages, monocytes	Patients with history of atopic dermatitis
Molluscum Contagiosum Virus	Epidermal keratinocytes	Children

Skin viruses, their cell tropism, and the populations vulnerable to infection		
Varicella Zoster Virus	Upper respiratory epithelium (primary infection), skin keratinocytes (hematologic transport via infected T cells)	<ul style="list-style-type: none"> • Children • Immunocompromised • Elderly (Herpes Zoster) • Males
Human Papillomavirus	Basal keratinocytes	Immunocompromised
Merkel Cell Polyomavirus	Keratinocytes, dermal fibroblasts	<ul style="list-style-type: none"> • Immunocompromised • Females

■ COMMON VIRAL SKIN INFECTIONS

Molluscum contagiosum

Molluscum contagiosum is caused by molluscum contagiosum virus (MCV), which is a virus of the Poxviridae family. In human skin, infection is localized in the epidermal layers, where it induces a typical, complex hyperproliferative lesion with an abundance of virus particles.

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It is characterized by numerous small, pink nodules, most often on the face, genitalia, or rectal area. The lesions can however also occur on other sites, like the back, arms, buttocks, and inner thighs.

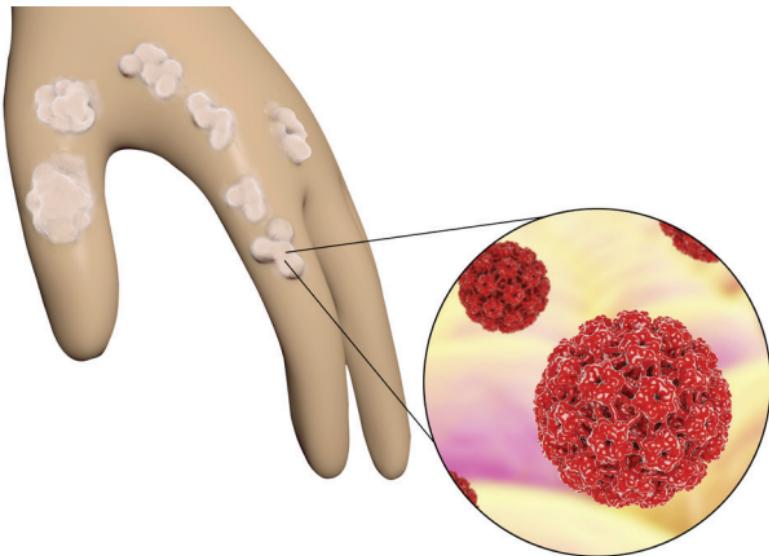
Close ups of Molluscum Contagiosum



Molluscum contagiosum is generally harmless and self-limiting, more commonly affecting preschool and elementary school-aged children. Skin lesions of molluscum contagiosum are called mollusca , typically presenting as asymptomatic, discrete, smooth, flesh-colored, dome-shaped papules with central umbilication from which a plug of cheesy material can be expressed. The duration of lesions is variable, though in many cases, they are self-limited in 6-9 months.

Papillomas (warts)

Papillomas are benign tumors arising from an epithelial surface, and usually known to grow in an outward direction; cutaneous papillomas are known as warts. Most of these lesions are caused by Human papillomavirus (HPV), of which there are over 100 types.

Illustration of hand with warts and close-up view of papillomavirus that causes development of wart

Besides skin, papillomas can however develop at other body sites with squamous epithelium also, e.g., lip, oral cavity, eyelid, and genital tract (genital warts). They are generally contagious upon contact with the exception of some cutaneous papillomas (skin tags) of the head and neck.

Risk factors in cases with non-genital warts include use of communal showers, occupational handling of meat, and immunosuppression.

Common types

1. *Verruca vulgaris* – These occur commonly on hands and fingers as single or multiple lesions, and are generally painless, firm, dry, and rough. They appear as slightly elevated, small plaques that are light brown or skin-coloured.

Wart on the hand finger



2. *Verruca plantaris* (plantar wart) – This clinical variety of warts occurs on the sole of the foot. These warts push into the skin during standing, walking, and running, and thus may be painful.

Plantar wart (verruca plantaris)

3. *Genital warts* (*condyloma acuminatum*) - appear as large lesions of red, soft masses, which may coalesce. They may be found in anal or genital area, including the penile shaft, scrotum, vagina, or labia majora. Color of the lesions is variable but tends to be skin-colored or darker, and they may occasionally bleed. Infection is highly contagious and spread through direct and sexual contact.

Condyloma acuminatum (Genital warts)

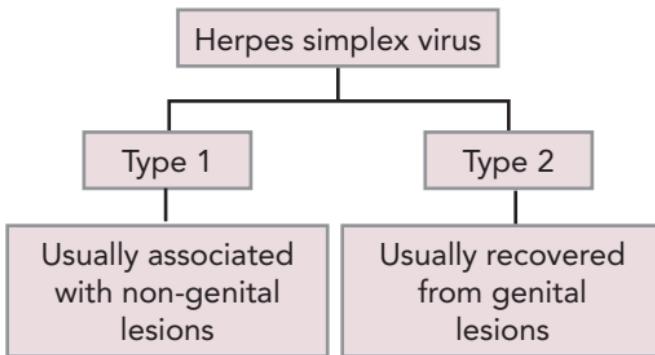


4. *Verruca plana juvenilis* (also known as juvenile flat warts) – Although they occur most commonly in children, adults may also get affected. The lesions occur in groups, and may appear on the face, neck, back of the hands, and arms.

Herpes simplex

Herpes simplex virus (HSV) infection is probably the most common viral skin disease, with a large reservoir in the general population. There are two types of HSV: Type 1 and Type 2.

Herpes simplex virus (HSV)



HSV infection can thus result in several mucocutaneous manifestations, e.g.:

- Gingivostomatitis
- Herpes genitalis
- Herpetic keratitis, and
- Dermal whitlows.

Transmission of HSV 1 is mainly oral, and that of HSV 2 is mainly genital. A critical factor for transmission of these viruses is the requirement for intimate contact between a person who is shedding virus and a susceptible host. After inoculation onto the skin/mucous membrane and an incubation period of 4 to 6 days, the virus replicates

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in epithelial cells. In sequence, cell lysis and local inflammation ensues with continuation of virus replication, thereby resulting in characteristic vesicles on an erythematous base.

Gingivostomatitis

Gingivostomatitis is usually caused by HSV 1, and occurs most frequently in children <5 years of age, typically affecting the tongue, lips, gingival, buccal mucosa and the hard and soft palate. The infection is characterized by fever and sore throat, followed by development of vesicular or ulcerative lesions on the oral and pharyngeal mucosa. Clinically, the skin lesions present as numerous pin-head vesicles, which rupture rapidly to form painful irregular ulcerations covered by yellow-grey membranes.

Herpes simplex labialis



Recurrent infections of the oropharynx most frequently manifest as herpes simplex labialis (cold sores), usually appearing as vesiculo-ulcerative lesions on the vermillion border of the lip.

Genital Herpes

Herpes genitalis can be caused by both type 1 or type 2 viruses, though type 2 is commonly the consideration in these cases. The infection can manifest as either a primary or recurrent infection.

A primary (initial) genital herpes infection in women usually involves the vulva, vagina, and cervix; and glans penis, prepuce or penile shaft in men. In both cases, the disease is associated with fever, malaise, anorexia, and bilateral inguinal adenopathy. In addition, women frequently complain of dysuria and urinary retention owing to urethral involvement. The complete healing of primary infection may take several weeks.

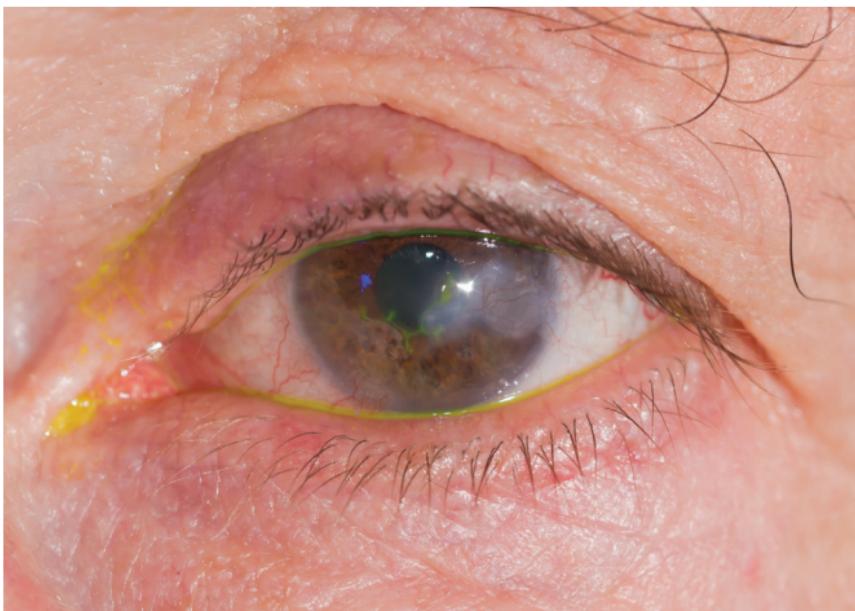
Recurrent genital infections can be seen in up to 2/3rd cases, and are particularly distressing. The frequency of recurrence however varies significantly amongst individuals.

Herpetic Keratitis

Herpes simplex keratitis, usually caused by HSV 1, is accompanied by conjunctivitis in many cases. The lesions of herpes simplex keratoconjunctivitis are dendritic ulcers. Involvement of deep stroma has been reported and may result in scarring and visual impairment.

The infection is primarily diagnosed by its clinical presentation, with common symptoms including redness, discharge, watery eyes, irritation, itching, pain, and photophobia. An urgent ophthalmological opinion should be sought for patients with ophthalmic zoster.

Herpetic Keratitis



Herpetic whitlows

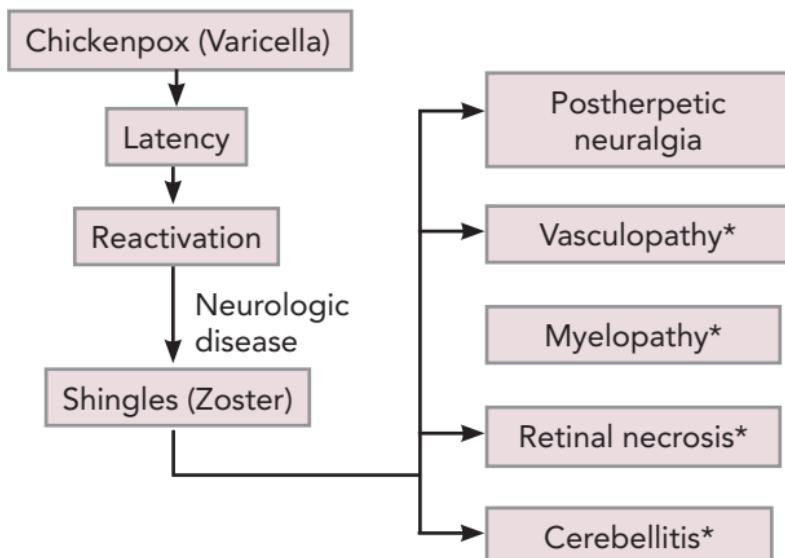
In general, HSV infections can manifest at any skin site. Herpetic whitlows, though rare in general, are common among healthcare workers, and manifest as lesions (vesicles) on abraded skin of the fingers, associated with pain, swelling, and erythema. It has also been documented in athletes who wrestle. The diagnosis of herpetic whitlow is typically clinical based on appearance of lesions and the patient's history. Infection usually involves just one finger, and vesicles usually coalesce into large, honeycomb-like bullae. Vesicular fluid is clear at the beginning but may become turbid, seropurulent, or hemorrhagic as it progresses.

Varicella-Zoster Virus

Two different manifestations of Varicella-zoster virus (VZV) can be seen dependent on whether it is a primary infection or that occurring due to reactivation of the latent virus. The virus is highly contagious, and is usually transmitted by droplets. In a susceptible individual, replication of virus in oropharynx leads to primary viremia, with successive development of a vesicular rash.

1. Primary VZV infection causes varicella (**chickenpox**)
2. Reactivation of latent virus (usually in adults) causes herpes zoster (**shingles**). This manifests as vesicular rash with a dermatomal distribution and acute neuritis.

Varicella-Zoster Virus infection



*May also develop without rash

Chickenpox

Chickenpox is the manifestation of primary VZV infection. It most commonly occurs in young children (1 to 9 years of age) and has a characteristic disseminated vesicular rash that appears after an incubation period of 14 to 17 days. Patients also have fever concurrently.

In children, however, the illness may not be preceded by prodromal symptoms, and the initial sign could be a rash, which typically begins on chest and face and then spreads to the extremities.

Morphologically, lesions begin as macules, rapidly progresses to papules, and then followed by a vesicular stage and crusting; crusts slough-off after 1 to 2 weeks.

Young Child with Chickenpox



Usually, in a patient there are always different stages of exanthema simultaneously, resulting in the picture of a “starry sky”. In a normal child, average duration of lesion formation is 3 to 5 days; though, duration is usually longer in adolescents and adults. The virus may establish latency in dorsal root ganglia at the time of primary infection.

Herpes zoster (shingles)

This is the recurrent form of VZV infection, which signifies a reactivation of the latent virus, and typically manifests as a localized vesicular rash with a dermatomal distribution.

The rash initially appears as unilateral erythema within the dermatome, and is soon followed by development of vesicles; in some individuals, vesicles may coalesce to form bullous lesions.

Development of rash is usually preceded by a prodromal phase characterized by pain, itching, paresthesias (numbness or tingling), dysesthesias (unpleasant sensations), or allodynia (sensitivity to touch) in one to three dermatomes.

New vesicles may form for 5 to 7 days, and then evolve through the sequence of healing. Average time to healing can range from 10 to 21 days, depending on individual's age and immune status.

The most common site affected is the chest, followed by lesions on the face. A characteristic of herpes zoster in most adults is the appearance of acute neuritis and post-herpetic neuralgia.

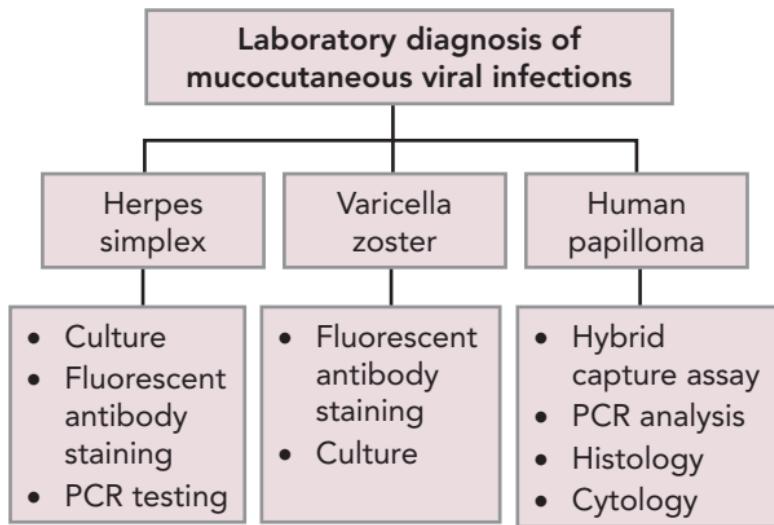
Herpes zoster (Shingles)



■ DIAGNOSIS OF VIRAL INFECTIONS

Skin lesions in most viral skin diseases are prominent and characteristic, and may therefore suggest a specific viral illness, the diagnosis of which can then be quickly established by appropriate laboratory methods like virus isolation, through culturing and/or on detection of viral genes or gene products. Stained smears of the vesicle fluid are often examined under the microscope for typical cytopathology and microbiologic diagnosis.

Laboratory diagnosis of viral infections of the skin and mucous membranes



Diagnosis of molluscum contagiosum

The diagnosis is based on clinical findings, while dermoscopy presents a useful clinical tool. Confocal microscopy or skin biopsy could be performed if there is diagnostic doubt.

Diagnosis of cutaneous warts

A patient with warts usually complains of a small rough bump arising from the skin that may either be painless or painful. There can be single or multiple solid papules. On the skin, the rough solid papule is often covered with hyperkeratinized skin, while those arising on mucosal

surfaces appear as a soft, pedunculated mass with several finger-like projections. The appearance of lesions may be affected by the extent of keratinization; while less keratinized lesions appear pink or red in color, heavily keratinized lesions appear white in color.

Most papillomas are thus diagnosed upon clinical examination, without requiring further investigations, especially in immunocompetent individuals. A biopsy may be necessary for confirmation, if there is uncertainty about the diagnosis or if the patient is immunocompromised.

Diagnosis of herpes simplex virus infections

The diagnosis of common herpetic infection can usually be based on clinical history and presenting features. Confirmatory laboratory diagnosis is required when patient may be immunocompromised. A definitive diagnosis requires either isolation of virus or detection of viral gene products. In addition, serologic assays may be used to help in distinguishing HSV infections.

Diagnosis of varicella

Diagnosis of varicella is based on characteristic vesicular rash/lesions. A laboratory diagnosis of acute VZV infection can be made by detection of the viral DNA using PCR in vesicle fluids. In most cases, vesicle fluids containing high virus load are suitable for viral isolation.

■ TREATMENT OF VIRAL INFECTIONS

Due to the limited number of effective antiviral agents, prevention appears to be important. Acyclovir (oral and intravenous) is effective for treatment of primary herpesvirus infection. Infections with HSV and VZV are amenable to antiviral therapy; acyclovir, valaciclovir and famciclovir.

Treatment of molluscum contagiosum

- Watchful waiting of lesions
- Active treatments (for cosmetic reasons or concerns of transmission and autoinoculation) may be:
 - » Mechanical (e.g., cryotherapy, curettage, pulsed dye laser therapy),
 - » Chemical (e.g., cantharidin, potassium hydroxide, podophyllotoxin, benzoyl peroxide, tretinoin, trichloroacetic acid, lactic acid, glycolic acid, salicylic acid),
 - » Immune-modulating (e.g., imiquimod, interferon-alpha, cimetidine), and
 - » Anti-viral (e.g., cidofovir).

Treatment choice often depends on the number and location of lesions, the prior experience of the treating physician, and the preferences of the patient or caretakers.

Treatment of cutaneous warts

Painless cutaneous papillomas may sometimes be left untreated and regress with time in immunocompetent individuals given the low potential for malignant transformation. Treatment, if indicated, varies depending on the type, size, and location of lesions. Cutaneous and genital warts are managed with topical medications or procedures, such as cryotherapy or laser surgery. In addition, skin warts may also be removed by excision. Topical salicylic acid increases cutaneous wart clearance compared with placebo. Though, papillomas often tend to recur around the primary site of infection, and may require retreatment. Oral zinc sulphate can be a therapeutic option for recalcitrant viral warts given its efficacy and tolerability.

Treatment of herpes simplex and varicella-zoster virus

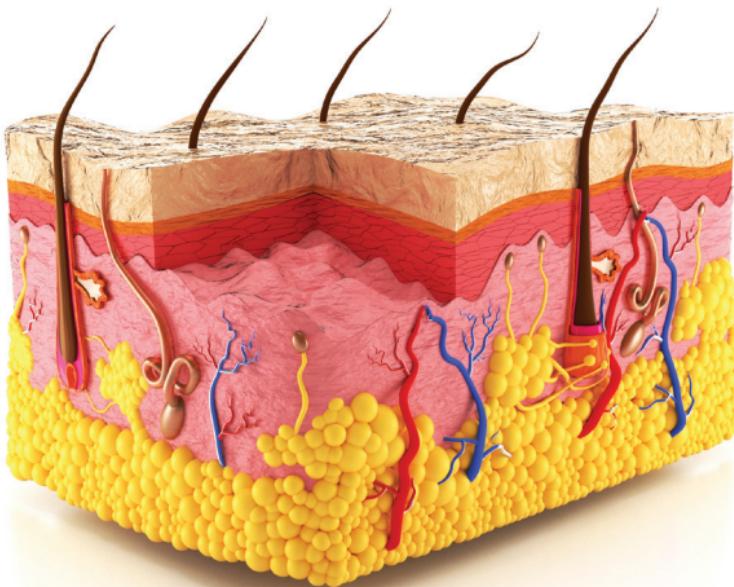
Infections due to HSV and VZV are amenable to therapy with antiviral drugs like acyclovir. Famciclovir and valaciclovir are also available for the treatment of zoster. Treatment is further supplemented with supportive care, like adequate fluid intake, antipyretics and analgesics (usually paracetamol), and good hygiene. Topical medications can be given for pruritic rash, while daily cleansing with warm water may help to avoid secondary bacterial infection.

Antiviral therapy considerations in herpesvirus and varicella virus infections			
Virus	Disease	Normal patient	Immuno-compromised patient
Herpes simplex	Primary	Oral acyclovir* 100–200mg, 5 times daily	Aciclovir 250mg/m ² intravenous every 8h
	Recurrent herpetic ulcers	Oral acyclovir* 100–200mg, 5 times daily	Aciclovir 250mg/m ² intravenous every 8h
	Recurrent herpes labialis	Topical Penciclovir 1%, or aciclovir 5% every 2h	Aciclovir 250mg/m ² intravenous every 8h

Antiviral therapy considerations in herpesvirus and varicella virus infections			
Herpes varicella	Chickenpox	-	Aciclovir 500mg/m ² (5mg/kg) intravenous every 8h
	Zoster (shingles)	3% aciclovir ophthalmic ointment for shingles of ophthalmic division of trigeminal	Aciclovir 500mg/m ² (5mg/kg) intravenous every 8h or famciclovir 250mg thrice daily
<p>Caution:</p> <p>Aciclovir: systemic preparations, caution in renal disease and pregnancy; occasional increase in liver enzymes and urea, rashes, central nervous system effects.</p> <p>Famciclovir: caution in renal disease and pregnancy; occasional nausea and headache.</p> <p>*In neonate, treat as if immunocompromised.</p>			

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PARASITIC INFECTIONS



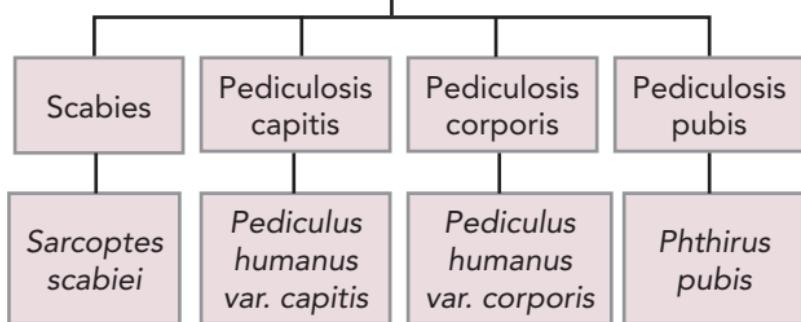
■ PARASITIC SKIN DISEASES

Parasitic skin diseases are frequent in human pathology, but often remain neglected. These infections affect individuals globally, though prevalence and burden is found to be especially high in subtropical and tropical countries. Some of the common risk factors for these parasitic skin diseases include lack of sanitation, crowding, and precarious housing conditions.

Amongst these infections, the two most common that are encountered in primary care are caused by ectoparasites; scabies or pediculosis. Host-parasite interactions in epidermal parasitic skin diseases are restricted to the stratum corneum, which is where ectoparasites complete their life-cycles.

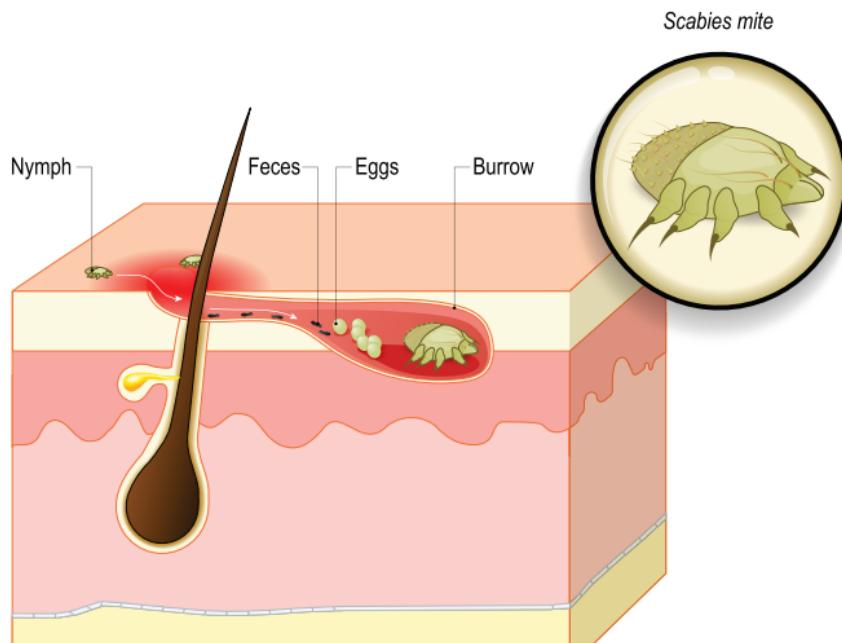
Biological agents of major epidermal parasitic skin diseases

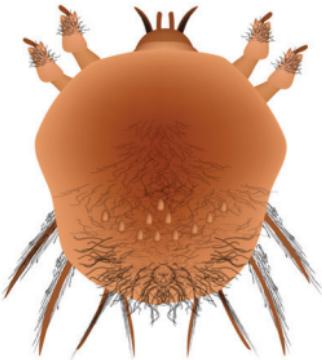
Parasitic skin diseases & their biological agents



Scabies

Scabies is caused by *Sarcoptes scabiei var. hominis*, which has humans as the only host. Transmission is mainly from person-to-person contact and only rarely through fomites. After exposure, mites can penetrate the epidermis within 30 minutes. The female mite penetrates the epidermis, digging furrows to lay eggs, and can release 40–50 eggs in 4 to 6 weeks.



Sarcoptes scabiei

back view



front view

A more severe clinical variety of scabies - **crusted scabies** (Norwegian scabies) - occurs when there is mite hyperinfestation; in fact, over one million parasites can be found in this variety.

Crusted scabies is observed primarily in immunosuppressed patients and is clinically characterized by intense pruritus and hyperkeratotic, crusted lesions with cutaneous fissures and thickened and dystrophic nails.

Scratching of the lesions can lead to secondary bacterial infections of the skin, such as impetigo, most commonly caused by *S. pyogenes* or *S. aureus*.

Crusted scabies**■ PEDICULOSIS (LICE)**

Pediculosis is an infestation of lice that can be further subdivided into following 3 types, dependent on the lice species that infest humans:

- Pediculosis capitis [caused by *Pediculus humanus capitis* (head lice)]
- Pediculosis corporis [caused by *Pediculus humanus humanus/corpus* (body lice)]
- Pediculosis pubis [caused by *Phthirus pubis* (pubic lice)]

Amongst these, head lice are the most common lice. These insects do not hop, jump, or fly, and are rather transmitted by person-to-person

contact. Eggs are attached to the hair at certain predilection sites.

Head lice



■ DIAGNOSIS OF PARASITIC SKIN DISEASES

Clinical signs demonstrated by patients with parasitic skin diseases may be related to the penetration of the parasite under the skin, its development, the inoculation of venom or allergic symptoms. Usually, patients with scabies and pediculosis present with itching as the primary symptom.

An elderly severely infested with scabies



Diagnosis of scabies

In most cases of scabies, intense pruritus, papular rash, and excoriations are characteristic; however, variable forms are possible. Diagnosis can be easy when clinical signs are pathognomonic (burrows in the interdigital web spaces appearing as short, wavy, scaly gray lines). Papules and burrows usually follow a characteristic distribution, with head and neck typically spared in adults; head and neck, however, may not be spared in infants and small children, and rash may include vesicles, pustules, or nodules.

SCABIES

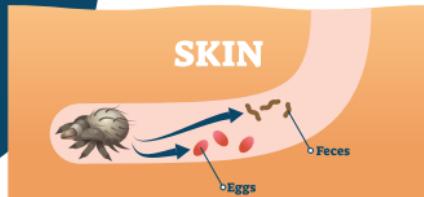
SCABIES is a SKIN INFESTATION caused by a MITE known as the *Sarcoptes scabiei*. Untreated, these microscopic mites can LIVE ON YOUR SKIN for months.



SCABIES MITE

The RASH itself can consist of tiny bites, hives, bumps under the skin, or pimple-like bumps.

The mites will burrow into the top layer of your skin to live and feed.



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Typically, infestations are seen under thin skin areas and concentrated especially in the body folds, like interdigital spaces, axillae, buttocks, and flexural surfaces of the wrists.

Scabies infestation in hand



Thus, diagnosis of scabies can often be made based on the combination of a history of intense pruritus (mainly at night), a classic rash, and itching in household contacts. Identification of mites on microscopic examination of skin scrapings obtained from the leading edge of the burrow and under the fingernails would be confirmatory.

Besides direct examination, dermoscopy (dermatoscopy) and epiluminescence microscopy can also be used for pathogen visualization.

Non-invasive techniques for scabies diagnosis		
Technique	Visualized structures	Required time
Dermatoscopy	Burrow	5-10 minutes (for fully examination)
Videodermatoscopy	Burrow, mite, eggs, larvae, faecal pellets	5-10 minutes (for fully examination)
Videomicroscope	Burrow, mite, eggs, larvae, faecal pellets	5-10 minutes (for fully examination)
Reflectance confocal microscopy	Burrow, mite, eggs, larvae, faecal pellets	~ 10 minutes (for each lesion*)
Optical coherence tomography	Burrow, mite, eggs, larvae, faecal pellets	~ 10 minutes (for each lesion)

*The handheld device allows real-time examination of each lesion.

Diagnosis of pediculosis

Similar to scabies, lesions of head lice are very itchy, causing the patient to frequently scratch its scalp; repeated scratching can often lead to excoriations, which facilitate bacterial superinfection.

A diagnosis of head and pubic lice infestations can objectively be made by finding lice or viable eggs on examination. Wet combing

is an accurate method to diagnose active head lice infestation, and visual inspection is the method of choice, if one aims to determine the frequency of carriers of eggs. Visualization can be further improved by the use of wood's light and dermatoscopy. Presence of a single live louse is adequate for the diagnosis of active infestation. Excoriations and pyoderma may also be present.

Infestation by body lice should be suspected when generalized itching occurs in persons who do not change or wash their clothing or bedding regularly; finding the lice in seams of clothing would be confirmatory. The infection causes pruritus of varying intensity, leading to erythematous macules, papules, scabs, and abrasions, mainly on the trunk, armpits, and buttocks.

■ TREATMENT OF PARASITIC SKIN DISEASES

Treatment of parasitic skin diseases includes both topical and oral agents. Especially, successful treatment requires strict adherence to directions for topical application.

Treatment of scabies

The treatment regime varies for each patient group, but general principles of treatment, like isolating the patient until completion of treatment, cleaning patient's room thoroughly, avoiding direct skin-to-skin contact and implementing strict hand-washing, machine-laundering patient's bedding and clothing separately using hot water, followed by hot dryer cycles, and prophylactic topical malathion 0.5% lotion prescribed to all household contacts or healthcare workers caring for patient, should be carried out diligently.

- *Permethrin 5% cream or lotion* - can be used in children, adults, pregnant women, and nursing mothers; should be applied over

entire body, from neck to toe after a bath; in children, it should also be applied to the scalp and retroauricular ridges. It should be applied at night-time, for 2 consecutive nights, and then on the 3rd day, in the morning, all bedding should be removed and washed.

- *Precipitated sulfur 5–10% in petroleum jelly* - can be used in children, pregnant women, and nursing mothers; should be applied on entire body for 4 consecutive nights, and removed during the day.
- *Oral ivermectin* - for adults and children > 5 years - 200 µg/kg, given as a single dose, which may be repeated after 7 days (ivermectin is not indicated in children < 5 years and pregnant women)
- *Ivermectin topical*, at 1%, in propylene glycol or as a lotion - applied on entire body; repeated application after 1 week
- 10% crotamiton; can be applied to scabies nodules in children
- *Crusted scabies*: dual therapy with oral ivermectin at 200 mcg/kg on days 1, 2, 8, 9, and 15, plus permethrin 5% cream full-body application daily for 7 days, then twice weekly until demonstrated cure.
- All clothing and bed linens should be changed.
- Decontaminate all linens, towels, and clothing used in the previous 4 days by hot-water washing (60°C) and heated drying, or dry cleaning.
- All household contacts should follow same cleaning procedures.

Head lice

- Wet combing
- *Permethrin shampoo 1%* - left on the scalp for 10 min and then rinsed
- *Piperonyl butoxide 15% shampoo*
- *Permethrin 5%* - applied to the scalp at night, and removed the next day; treatment should be repeated after 7 to 10 days.

- *Malathion 0.5%* - a 2nd-line agent effective in treatment of resistant head lice infestation; should not be used in neonates and infants, nursing mothers, and children <6 years
- *5% benzyl alcohol in mineral oil*; two 10-min applications should be performed, with a 7-day interval; can be used from 6 months of age onwards
- *Dimethicone 4% lotion/gel* - should be applied for 10 to 15 min; the lotion, for 8 h; application should be repeated in 7 to 10 days.
- *Ivermectin topical 0.5% lotion* - for children aged 6 months or older - should be applied for 10 min, and rinsed immediately after
- *Oral ivermectin 200 µg/kg* - Single-dose; repeated after 7 to 10 days.

Resistance should be suspected if live lice are still present 12 to 24 hours after treatment and no other cause for failure can be found. These cases should be treated with an agent from a different insecticide class.

Pubic lice

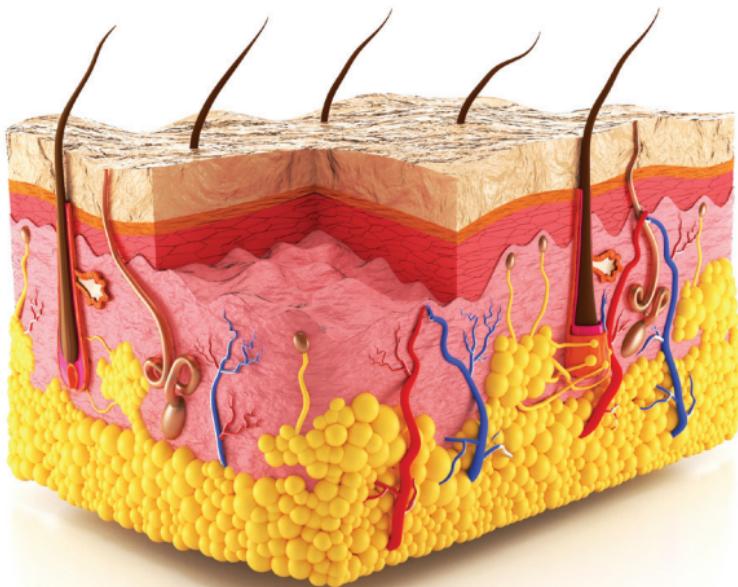
- Treatment generally same as head lice
- *Permethrin 5% or deltamethrin 0.02% cream* - applied at night and removed the following day; should be used for 2 consecutive days, and repeated after 7 to 10 days

Body lice

- Ask patients to wash entire body thoroughly and then put on clean clothing
- *Topical permethrin, pyrethrin, or malathion* in case of sever infestation
- *Oral ivermectin* (an alternative to topical treatment)
- Decontaminating clothing and bedding by hot-water washing (60°C) and heated drying, or by dry cleaning.

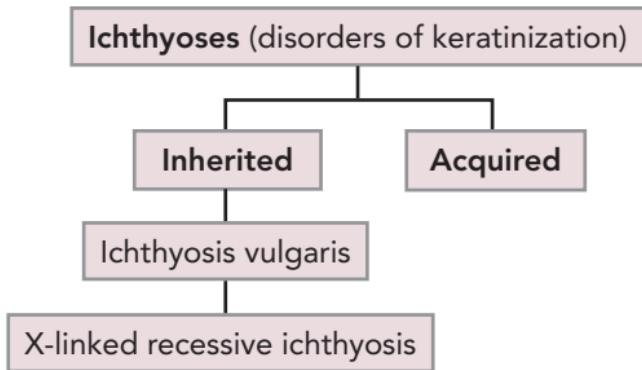
SECTION 8

ICHTHYOSIS & KERATODERMAS



■ ICHTHYOSIS

The term “ichthyosis” - coming from the Greek, meaning fish - is used to describe continual and widespread scaling of the skin, characterized by dry, thickened, scaling skin, and hyperkeratosis. It may be inherited (genetic) or acquired during life. There are several recognized forms of ichthyosis and related skin types, but most varieties are relatively rare, with some exceptions like ichthyosis vulgaris and the X-linked recessive ichthyosis.



■ ICHTHYOSIS VULGARIS

Ichthyosis vulgaris is one of the more commonly seen types of ichthyosis that appears in approximately 1 in 250 individuals. The condition frequently goes undiagnosed in practice because people often think it as simple “dry skin” and never seek treatment.

Skin pathology

In patients with ichthyosis vulgaris, skin cells are produced at a normal rate; however, they do not separate normally at the surface of *stratum*

corneum and are not shed as quickly as they should be, thereby resulting in a build-up of scales.

Clinical presentation

Clinical symptoms of ichthyosis typically present at birth or within the first few years of life. Often, the condition is mild, and only a portion of the body may be involved, with scaling (fine, light grey scales) and roughness most common and most severe in lower limbs. Face usually remains unaffected, and when affected, scaling is usually limited to the cheeks and forehead. Ichthyosis vulgaris is more obvious during winter time, and may improve during the summer.

Ichthyosis vulgaris



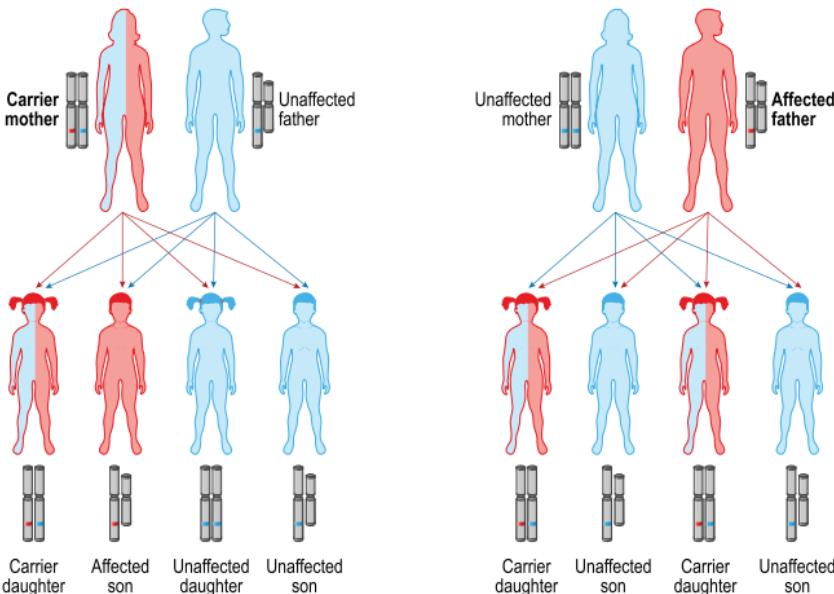
Treatment

- Topical moisturizers (most effective when applied on wet skin within a couple of minutes of having a shower/bath)
- Topical keratolytics (should be used with caution if over large body surfaces)

■ X-LINKED ICHTHYOSIS

X-linked ichthyosis is another more commonly seen form of ichthyosis that occurs in approximately 1 in 6,000 births. The condition occurs only in males, and can range from mild to severe. Skin findings usually appear within the first year of life, with 15% to 20% having manifestations at birth.

X-linked recessive inheritance



Skin pathology

Generally same as ichthyosis vulgaris

Clinical presentation

The scales of X-linked ichthyosis are often dark (tan or grey) and usually cover only a portion of the body. Typically, the back of the neck is almost always affected, while face, scalp, palms and soles are spared. Similar to ichthyosis vulgaris, X-linked ichthyosis frequently shows improvement in the summer. Pruritus usually is absent.

Treatment

- Topical moisturizers (most effective when applied on wet skin within a couple of minutes of having a shower/bath)
- Topical keratolytics (best avoided in first 6 months of life and should be used with caution if over large body surfaces)
- Cholesterol containing ingredients may improve scaling.

Other less common inherited ichthyoses

- Non-bullous ichthyosiform erythroderma
- Lamellar ichthyosis
- Harlequin ichthyosis
- Netherton's syndrome
- Epidermolytic ichthyosis

■ PALMOPLANTAR KERATODERMAS

Palmoplantar keratodermas comprise a diverse group of disorders marked by excessive (hyperkeratotic) thickening of the epidermis of palms and soles; which develops as a compensatory hyperproliferation

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of the epidermis and excessive production of *stratum corneum* in response to altered cornification of the palmoplantar skin. These can be acquired, but are mainly hereditary and attributable to a plethora of mutations in many genes.

Palmoplantar keratoderma



Some possible causes of acquired Palmoplantar keratodermas

- Exposure to certain chemicals (e.g., arsenic, chlorinated hydrocarbon fluids)
- Side effects of certain drugs (e.g., beta-glucan, lithium, chemotherapy agents)
- Metabolic disorders (gravidity, menopause, hypothyroidism, myxedema)

Clinical features

Usually, an early onset and positive family history suggest a genetic cause for palmoplantar keratoderma. Clinically, though, a morphological distinction can be made between diffuse/plane, focal (patchy, striate, filiform, or discoid) or punctate with small, round hyperkeratotic lesions.

Besides; the severity of disease, involvement of areas other than the palms or soles, and the onset of other symptoms, perhaps as part of a syndrome, are useful to classify/diagnosis the disorder.

Classifying palmoplantar keratoderma based on extent of involvement

- *Isolated palmoplantar keratodermas* - thickening of the skin of palms and soles is the main manifestation
- *Syndromic palmoplantar keratodermas* - include other ectodermal defects and/or extracutaneous manifestations in addition to palmoplantar involvement

The most notable histological feature of acquired palmoplantar keratosis is hyperkeratosis of the *stratum corneum*. Another possible sign of the acquired form is symptoms that are limited to the palms and soles.

Clinical manifestations of hereditary palmoplantar keratoderma usually begin at birth or soon thereafter; herein, as mentioned earlier, exact morphological evaluation of keratoderma is diagnostically helpful. Hyperhidrosis can be common on the feet, frequently predisposing to bacterial and fungal infections.

Different clinical-morphological aspects of keratoderma	
Diffuse	Affecting the entire surface of palms and soles
Transgradient	Lesions extend beyond palmoplantar skin
Cicatrizing or mutilating	With constricting bands around digits
Focal or striate	Areas of palmoplantar skin most exposed to pressure are disproportionately thickened
Punctate	With multiple scattered discrete round lesions

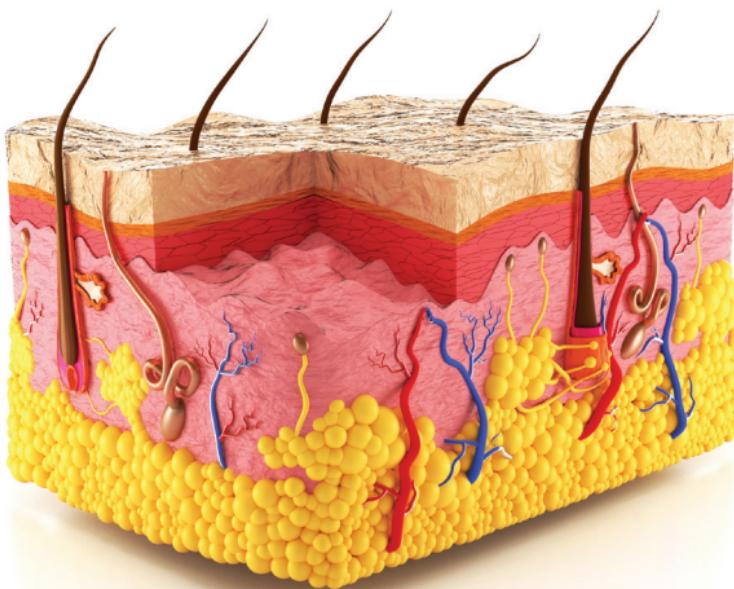
Treatment of palmoplantar keratoses

The choices of treatment are generally individualized, and accompany topical antibacterial and antifungal prophylaxis also.

- Treat/eliminate cause (in patients with acquired disease); e.g., toxins, infection, other factors
- Rehydration and skin care
 - » Regular baths (to cleanse and hydrate areas of keratinization)
 - » Topical moisturizer (to ensure optimal hydration of skin)
 - » Topical therapy with urea-based ointments
 - » Topical vitamin D
 - » Mechanical keratolysis (as needed)

SECTION 9

MISCELLANEOUS DISORDERS (URTICARIA/ PRURITUS)



■ URTICARIA

Urticaria, also known as “hives”, is a common, mast cell-driven disease presenting with wheals or angioedema, or both. Histamine and other inflammatory mediators, such as leukotrienes and prostaglandins, are considered major players in the development of symptoms of urticaria.

Urticaria is classified into different subgroups mainly based on the clinical criteria: acute and chronic urticaria. Acute urticaria is defined by a repeated appearance of wheals with/without angioedema over a period that last < 6 weeks, whereas recurrence of lesions over > 6 weeks is considered as chronic.

Urticaria (hives) on the back



Etiology

Acute urticaria may generally be associated with identifiable causes, like an acute viral infection or an allergic reaction to medications, insects or foods. Alternatively, chronic urticaria can be further subdivided into two groups: chronic spontaneous urticaria (previously termed chronic idiopathic urticaria) and chronic inducible urticarial that includes both physical and non-physical urticarias. Anxiety, obesity, dissociative and somatoform disorders, and malignancies are associated with an increased risk to develop chronic spontaneous urticaria.

Chronic inducible urticarias

Physical

Symptomatic
dermographism/
urticaria factitia

Cold- and
heat-induced
urticarias

Delayed
pressure
urticaria

Solar urticaria

Vibratory
angioedema

Non-physical

Cholinergic
urticaria

Contact
urticaria

Aquagenic
urticaria

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Some distinguishing features of chronic spontaneous urticaria from chronic inducible urticarial		
Chronic urticaria	Diagnosis	Duration of lesions
Chronic inducible urticaria	History of a provocative stimulus - (cold, heat, pressure, solar, etc.) Challenge by a challenge test to confirm suspected stimulus - <i>ice cube test</i>	Most often lesions short-lived (minutes to 2 hours)
Chronic Spontaneous urticaria	History identifies a lack of a consistent trigger for appearance of the majority of lesions Pressure is a common stimulus reported	4 to 24 hours

Clinical presentation

Urticaria is marked by onset of pruritic “wheals,” which represent well-circumscribed areas of non-pitting edema with blanched centers and raised borders that involve only the superficial portions of dermis and are seen together with surrounding erythema of the skin. The size of lesions can be of few millimeters in diameter, but they can coalesce to form wheals as large as several centimeters wide.

Urticaria on skin



SECTION 9

Angioedema, at submucosal surfaces of upper respiratory and gastrointestinal tracts and deeper layers of skin including subcutaneous tissue, may also be present. The onset of symptoms is rapid.

Wheal vs. Angioedema	
Wheal	Angioedema
<ul style="list-style-type: none">Characterized by a circumscribed superficial edema of the skin, mostly surrounded by a bright red erythema and associated with a strong itching or burning sensationDevelop within several minutes and have a transient nature (1-24 hr)	<ul style="list-style-type: none">Presents as painful or burning, non-itchy, and less well-demarcated edema of the deep dermis and subcutis, or mucous membranesUsually, appears as skin-colored or slightly red swellingsDevelop slowly and may persist for several days

Diagnosis

The diagnosis of urticaria is usually clinical.

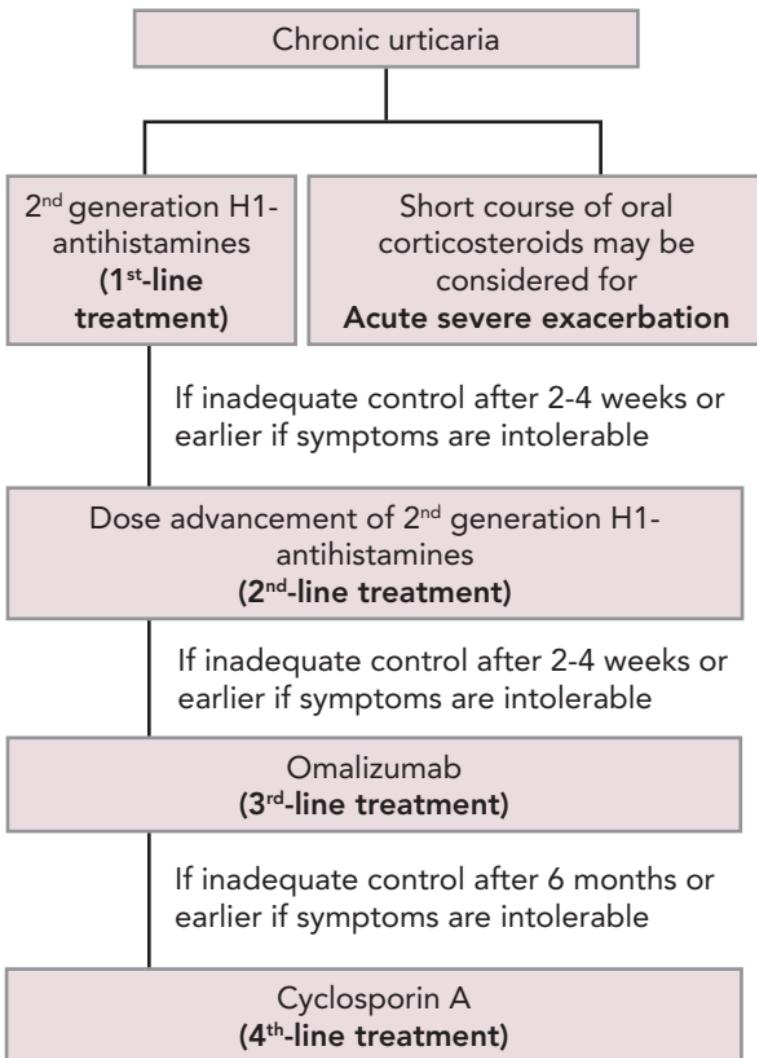
- A thorough history, with information on onset, frequency and duration of symptoms and of individual urticarial lesions (lasting >24 hours would suggest vascular component rather than an IgE-mediated phenomenon)

- History should include information about medication and supplement use, allergies, recent infections, travel history, and any systemic illnesses.
- If patient report identifiable triggers for chronic inducible urticaria; perform specific provocation and threshold testing.
- If chronic spontaneous urticaria is suspected, initial blood work should include complete blood count with differential and ESR or CRP
- Omission of suspected drugs (e.g., NSAIDs)

Treatment

- Avoid wearing tight clothing
- Avoid identified triggers
- 2nd-generation non-sedating H1-antihistamines, e.g., bilastine
- Omalizumab (an anti-IgE antibody)
- Short course of an oral corticosteroid (prednisolone 0.5 to 1 mg/kg/day for 3 to 10 days) - for treating acute exacerbations; long-term use should be avoided)
- Angioedema of larynx and massive angioedema of the tongue are medical emergencies owing to the risk of airway obstruction.

A management algorithm for chronic urticaria



■ PRURITUS

Pruritus, or itch, is a common and unpleasant sensation that evokes the urgent desire to scratch, and is often the most common symptom of skin diseases. It represents a complex process that commonly occurs with several skin disorders, and involves various cells, mediators and receptors in the peripheral skin and the central nervous system. Acute pruritus lasts < 6 weeks, whereas chronic pruritus persists for ≥ 6 weeks and can often be difficult to treat.

A person with itch on upper limb



Etiology

Besides common dermatologic causes such as urticaria and atopic dermatitis, pruritus could also be a symptom related to an underlying disease process such as cholestasis or hyperthyroidism, or simply be caused by dry skin, especially in cold, winter months.

Underlying etiologies of pruritus	
Etiology	Examples
Dermatologic	Atopic dermatitis, psoriasis, urticaria, lichen planus, bullous pemphigoid
Systemic	Chronic kidney disease, cholestatic diseases of the liver, lymphoma, drug-induced pruritus without rash, hyperthyroidism
Neurologic	Brachioradial pruritus, notalgia paresthetica, multiple sclerosis
Psychogenic	Obsessive-compulsive disorder, depression, anxiety disorders, delusions of parasitosis
Mixed	Chronic pruritus with xerosis in a patient with cholestatic disease
Undetermined	Chronic pruritus of unknown origin, mature aging itch

Diagnosis

The first step in diagnosis of pruritus requires identification of the underlying cause, which may be dermatologic or non-dermatologic

in nature. The process thus begins with a thorough history consisting of past medical, family, and drug history, and physical examination, followed by laboratory tests and imaging if required. Attention must be paid to the onset, nature, duration, location, severity, relationship to activities, time relation, and precipitating, alleviating, and exacerbating factors.

The location of itch may provide some cues, for instance, neuropathic pruritus is likely to manifest as localized pruritus (often along a dermatome), drug-induced pruritus more likely presents as generalized itching, while pruritus of cholestasis often initially appears on palms and soles. Furthermore, during physical examination, primary skin lesions should be distinguished from lesions secondary to chronic scratching.

Description of itch	
Onset	Sudden or gradual
Nature	Continuous, intermittent, burning, pricking
Duration	Minutes, hours, environmental days, weeks
Severity	Does it interfere with normal activity
Time relation	Bedtime, cyclical
Location	Localized, generalized, unilateral or bilateral, scalp
Relationship to activities	Worse at work or when doing hobbies
Provoking factors	Heat, exercise, water
Alleviating factors	Distraction, antihistamines, cool compresses

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Systemic signs and symptoms such as weight change, night sweats, fever, chills, abdominal pain, fatigue, myalgias, lymphadenopathy, and changes in bowel pattern should prompt further investigation for systemic disease, given the fact that a systemic etiology is possible in 16-50% of patients with generalized pruritus.

Some dermatologic signs of chronic scratching

- Excoriations (i.e., scratch marks),
- Lichenification (i.e., areas of thickened skin resulting from repeating rubbing),
- Prurigo nodularis (i.e., nodular lesions), or
- A combination of these.

Treatment

Treatment of pruritus is often aimed at eliminating the underlying cause first, followed by management of the itchy sensation, based on the diagnosis, severity of itch, and individual patient preference. Treatment may include topical and systemic medications, hydrotherapy, phototherapy, and ultraviolet therapy.

Topical treatment options for pruritus

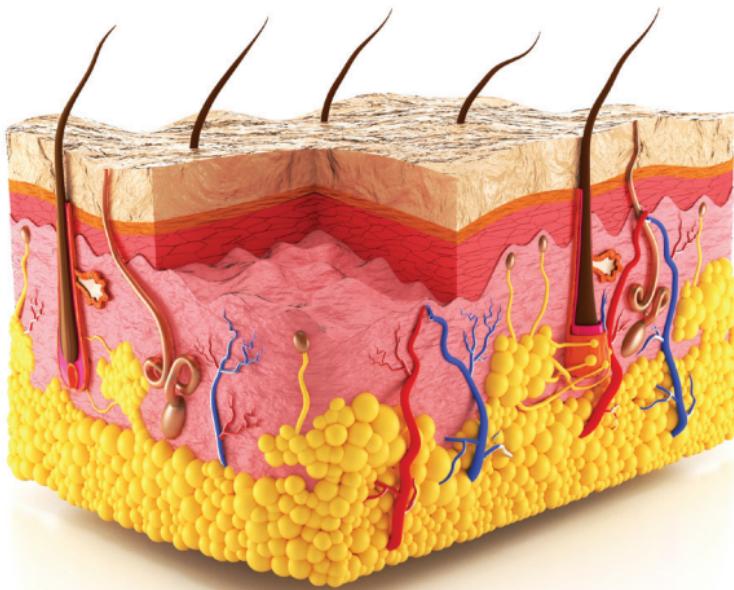
- Topical emollients or moisturizers
- Topical corticosteroids (should not be used on large body surfaces and for long periods of time)
- Topical calcineurin inhibitors (tacrolimus and pimecrolimus)
- Topical anesthetics (ketamine, amitriptyline, and lidocaine); Topical cooling agents, such as menthol, camphor, and phenol; Topical cannabinoids

Miscellaneous Disorders (Urticaria/Pruritus)

Systemic therapies for treatment of pruritus				
Class of drugs	Examples	Dose	Frequency	Indication for use
Antihistamines	Bilastine	20 mg	od	Urticaria, pruritus
	Levocetirizine	5 mg	od	
	Diphenhydramine	25-100 mg	qid	Urticaria, nocturnal itch
	Doxepin	10-50 mg	1-3 times daily	
Anticonvulsants	Gabapentin	100-1200 mg	tid	Neuropathic itch, pruritus or unknown origin
	Pregabalin	25-200 mg	bd	
Opioid modulators	Butorphanol	1-3 mg/mL	At bedtime	Intractable itch
	Naltrexone	25-50 mg	od	Cholestatic pruritus
Antidepressants	Sertraline	75-100 mg	od	Cholestatic pruritus
	Paroxetine	10-40 mg	od	Psychogenic pruritus, paraneoplastic pruritus
	Fluvoxamine	25-150 mg	od	
	Mirtazapine	7.5-15 mg	At bedtime	Nocturnal itch

SECTION 10

ECZEMA & DERMATITIS



■ ECZEMA & DERMATITIS

Eczema and dermatitis are both generic terms for skin inflammation that are often used interchangeably to describe a variety of skin conditions consisting of red, dry patches of skin and rashes.

Particularly, however, the term eczema is used interchangeably for “atopic dermatitis” and not for other types of dermatitis.

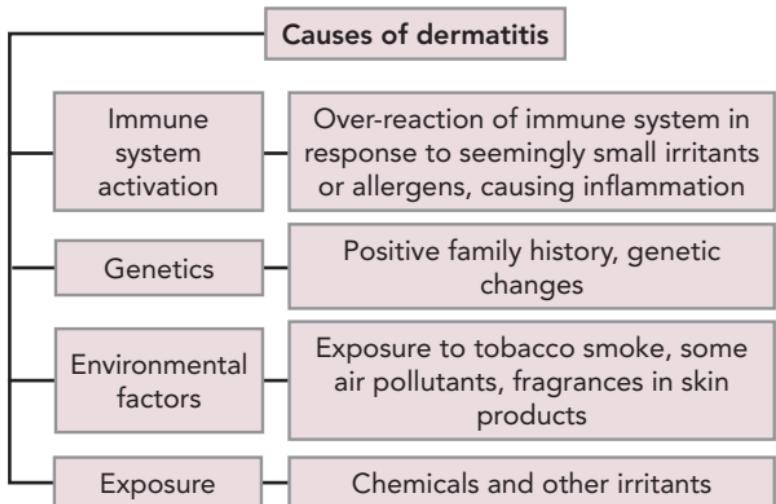
■ TYPES OF DERMATITIS

Several types of dermatitis forms can be seen in clinical practice, the common of which include atopic dermatitis (eczema), contact dermatitis, and seborrheic dermatitis. Some other types of dermatitis include, but are not limited to:

- Diaper dermatitis
- Nummular dermatitis
- Dyshidrotic dermatitis
- Perioral dermatitis
- Stasis dermatitis

Causes of dermatitis

Dermatitis can be caused by a combination of several factors that induce skin barrier dysfunction together with cutaneous and systemic immune dysregulation.



Common signs and symptoms

The signs and symptoms in patients with dermatitis usually depend on the type of dermatitis, and each type may have varying combinations of common skin symptoms.

Common signs and symptoms of dermatitis

- Itching and red rashes
- Dry skin
- Fluid-filled blisters
- Thickening, hardening and swelling of skin
- Crusting, scaling and creasing skin
- Painful ulcers
- Rashes may ooze fluid or bleed when scratched.

■ COMMON PATTERNS/TYPES OF DERMATITIS

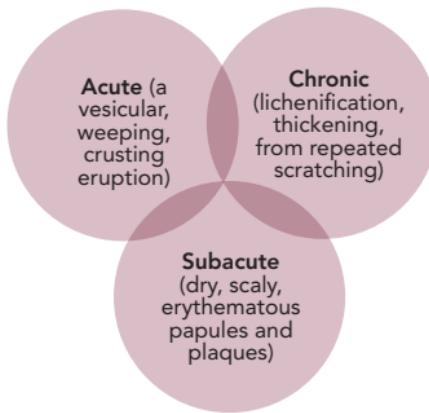
Atopic dermatitis (eczema)

Atopic dermatitis, or atopic eczema, is a chronic and relapsing inflammatory skin disease that often develops during childhood, and has a wide spectrum of signs and symptoms contributing towards profound functional disturbances. A positive family history of atopic diseases is the strongest known risk factor, and the disease is commonly associated with other atopic manifestations such as food allergy, allergic rhinitis, and asthma; with onset most common by 5 years of age.

Clinical features

Atopic dermatitis is considered a lifelong disease with highly variable clinical phenotypes, but is often characterized by acute flare-ups of eczematous, oozing or weeping pruritic lesions over dry skin. Chronic lesions include red/brownish patches of dry, cracked or scaly skin with lichenification and prurigo nodules.

Clinical phases generally seen in atopic dermatitis



A child with dermatitis on face



Atopic dermatitis on legs



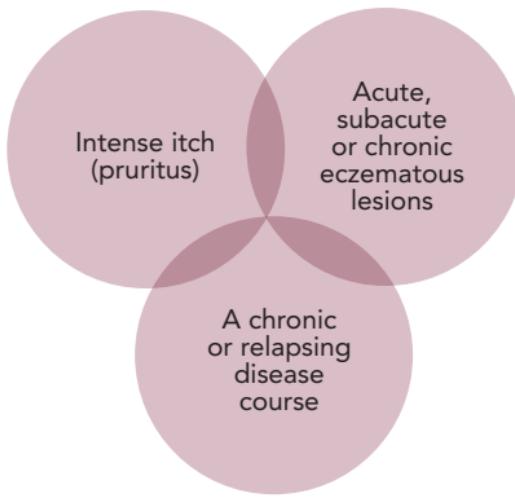
Atopic dermatitis at hand



Diagnosis of atopic dermatitis

Diagnosis of atopic dermatitis is based on specific criteria, which consider patient and family history and clinical manifestations. In all, disease severity needs to be determined by evaluating both subjective and objective signs and symptoms. Herein, some commonly used tools for assessing the disease severity include Eczema Area and Severity Index (EASI) and SCORing Atopic Dermatitis (SCORAD).

Essential features for diagnosis of atopic dermatitis



The lesions of atopic dermatitis can occur on any part of the body, though typically showing an age-related distribution pattern. Morphological subtypes of atopic dermatitis include:

- a. The follicular type, and
- b. The chronic prurigo type.

Common distribution pattern of atopic dermatitis lesions in different age-groups	
Infants	<ul style="list-style-type: none"> Often widely distributed and more-acute skin lesions characterized by severe erythema, edema, excoriations and serous exudate manifesting as oozing and crusting Characteristically located on the face/cheeks and trunk, with sparing of diaper area
Children	<ul style="list-style-type: none"> More localized and chronic with paler erythema, xerosis Skin thickened from repetitive scratching, seen mostly over the folds, bony protuberances and forehead Commonly affect flexor surfaces (antecubital and popliteal fossae, and buttock-thigh creases)
Adolescents and adults	<ul style="list-style-type: none"> A diffuse pattern but also with localized lesions, most typically affecting hands and flexures Adults can present only with chronic hand involvement or the head-and-neck subtype, which involves upper trunk, shoulders and scalp

Morphological subtypes of atopic dermatitis	
Follicular type	<ul style="list-style-type: none"> Characterized by densely aggregated follicular papules Frequent in dark-skinned individuals and people of Asian origin
Chronic prurigo type	<ul style="list-style-type: none"> Characterized by erythematous, often excoriated papules and indurated nodules Sometimes seen in patients with long-standing disease

Treatment of atopic dermatitis

Treatment goals in patients of atopic dermatitis require focusing on reducing pruritus and establishing disease control, with selection of therapy based on disease severity, patient's age, and comorbidities.

In general, patients should be advised on basic skin care and avoidance of triggers, and use of topical anti-inflammatory agents should be considered in disease flares or chronic/recurrent lesions.

In cases having an inadequate response, options like phototherapy, systemic immunosuppressants and biologic agents (e.g., dupilumab, a monoclonal antibody) are available. Treatment of moderate-to-severe atopic dermatitis though remains a challenge, requiring more targeted treatments.

Treatment for atopic dermatitis		
Atopic dermatitis severity	Treatment	
	Children	Adults
General (basic) therapy	Education, emollients, bath oils, and avoidance of clinically relevant allergens	
Mild (or transient eczema)	Reactive therapy with topical glucocorticosteroids class II, or depending on local cofactors (topical calcineurin inhibitors, antiseptics including silver)	Reactive therapy with topical glucocorticosteroids class II, or depending on local cofactors (topical calcineurin inhibitors, antiseptics including silver)
Moderate (or recurrent eczema)	Proactive therapy with topical tacrolimus or glucocorticosteroids class II, wet wrap therapy, psychosomatic counseling, climate therapy	Proactive therapy with topical tacrolimus or glucocorticosteroids class III, wet wrap therapy, UV therapy, psychosomatic counseling, climate therapy

Combination treatment with topical glucocorticosteroid (clobetasol) + zinc sulphate may provide more encouraging results than glucocorticosteroid (clobetasol) alone cream in the treatment of chronic hand dermatitis/eczema.

Treatment for atopic dermatitis		
Severe (or persistent eczema)	Hospitalization, systemic immunosuppression (cyclosporine A, methotrexate, azathioprine)	Hospitalization, systemic immunosuppression (cyclosporine A, short course of oral glucocorticosteroids, methotrexate, azathioprine, mycophenolate mofetil)
Additional treatment options should be considered for every phase; antibiotics/antiseptics should be added in case of superinfection; compliance and alternate diagnosis should be considered if therapy has insufficient effect.		

Contact dermatitis

Contact dermatitis – as the name suggests – occurs as a consequence of contact with various irritants or allergens. The disease can be caused by chemicals or metal ions that exert toxic effects without inducing a T-cell response (contact irritants), or by small reactive chemicals that induce an immune responses (contact allergens). A majority of cases of contact dermatitis are usually self-limited and managed with simple supportive measures; though it may become chronic in some patients. Contact dermatitis is further subdivided into following two types:

1. Irritant contact dermatitis

- A nonspecific response of skin to direct chemical and/or physical damage that releases mediators of inflammation primarily from epidermal cells; no prior exposure to the substance (sensitization) is required.

- b. Mainly causes skin barrier disruption, epidermal cellular changes, and cytokine release.
- c. Sufficient inflammation arises from the release of pro-inflammatory cytokines from keratinocytes, in response to the stimuli.
- d. The likelihood increases with the duration, intensity, and concentration of the irritant.
- e. Reaction usually reaches its peak quickly, within minutes to few hours after exposure, and then starts to heal.

2. Allergic contact dermatitis

- a. A delayed (type 4) hypersensitivity reaction to exogenous contact antigens in sensitized individuals.
- b. Common etiological allergens are nickel, chromium, neomycin, formaldehyde, paraphenylenediamine, fragrance mix, cobalt, poison Ivy.
- c. Reaction elicitation time depends on the sensitizer's characteristics, intensity of exposure, and the degree of sensitivity.
- d. Usually, lesions appear 24 hours after exposure to causative agent and reach their peak at ~72 hours.

Clinical presentation

Symptoms of irritant contact dermatitis may include the following, particularly at the beginning of the clinical course: burning, itching, stinging, soreness, and pain. Pruritus, though, is more common in allergic contact dermatitis. Major clinical differences between irritant and allergic contact dermatitis, however, include the more rapid onset of irritant contact dermatitis and the tendency of allergic contact dermatitis to spread.

Irritant contact dermatitis

Irritant contact dermatitis can occur as an acute or chronic disease. Lesions may occur anywhere but commonly appear on hands. Acute irritant contact dermatitis is typically characterized by erythema, blisters, pustules, hemorrhage, crusts, scales and erosions, and also with pruritus or even pain. Skin lesions here are predominantly sharply bordered in areas of contact.

Irritant contact dermatitis



Conversely, chronic irritant contact dermatitis is characterized by diffuse/localized lesions with poorly defined erythematous scaly patches and plaques, dryness of skin, lichenification and desquamation.

Irritant skin lesions commonly occur on the back of hands and forearms. Further, as the disease persists, lichenification and fissures develop, with possible nail damage.

Allergic contact dermatitis

In allergic contact dermatitis skin lesions, different clinical phases can be seen, i.e. erythematous phase with an unsharply delineated erythema or skin edema; and *madidans* phase characterized by erosions and moistening. In the next stage, crusts appear, followed by the final squamous stage, when the horny layer repairs itself.

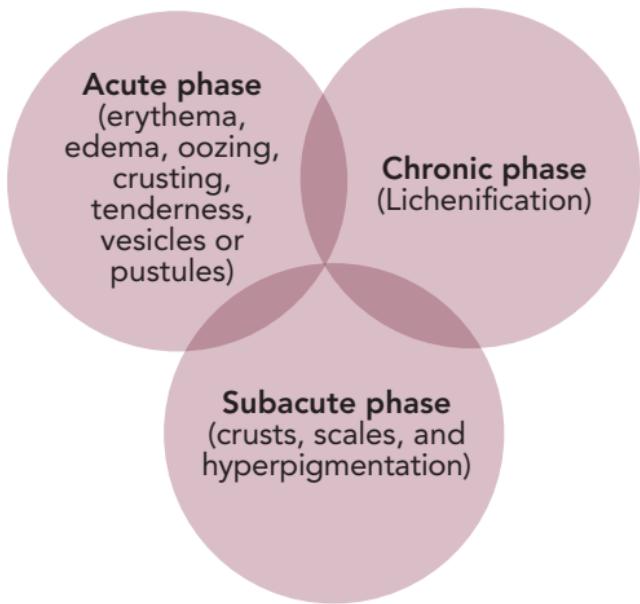
Allergic contact dermatitis at hand



Allergic contact dermatitis at neck

Acute allergic contact dermatitis develops after 24-48 hours. Skin lesions are initially asymmetric and limited to area of contact, but often disseminate later. Swelling and blistering can be seen in case of severe reactions. When skin lesions persist, chronic allergic contact dermatitis can develop; main feature of chronic allergic contact dermatitis is epidermal reaction with lichenification, fissures, and pruritus.

Morphological patterns possible in contact dermatitis



Diagnosis

Diagnosis of contact dermatitis depends on both history and clinical evaluation. History should include questions about occupation, hobbies and use of any topical or oral medications. Patch testing is considered a gold standard in diagnosing contact allergic dermatitis, and is useful in determining the exact cause when used in correlation with details of exposure and physical examination.

Treatment

- Avoidance of causative allergen (wear appropriate clothing to protect against irritants)

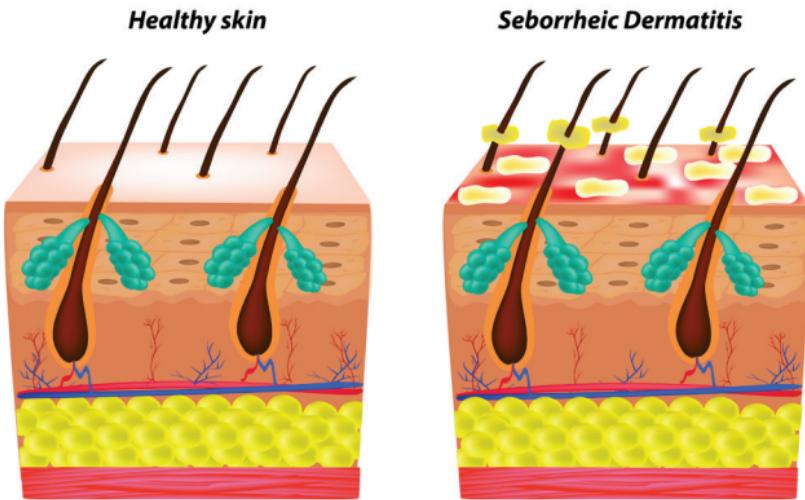
- Avoid use of soaps, perfumes, and dyes
- Emollients - for skin hydration
- High-potency topical corticosteroids, e.g. clobetasol propionate (0.05% cream) - to reduce inflammation (should not be used on thin skin, e.g. face, genitals, intertriginous areas to avoid risk of skin atrophy)
- Antihistamines, such as bilastine and levocetirizine - to control pruritus
- Systemic steroids - in severe cases (should be tapered gradually to prevent recurrences)
- Immunomodulating drugs like tacrolimus ointment and pimecrolimus cream - helpful in allergic contact dermatitis.

■ SEBORRHEIC DERMATITIS

Seborrheic dermatitis is an inflammatory, papulosquamous skin disease that affects areas rich in sebaceous glands, e.g., the scalp, face, and body folds. The disease is most prevalent in infancy and middle age, but usually presents differently in these two age groups. A non-inflammatory variant of seborrheic dermatitis is commonly termed as dandruff.

In infants, the disease often appears as firm, greasy scales on the crown and frontal regions of scalp, in the first 3 months of life and is mild, self-limiting, resolving spontaneously in most cases by the 1st year of life. In contrast, the adult variant is characterized by a relapsing and remitting pattern of disease.

Seborrheic dermatitis



Etiology

The onset of seborrheic dermatitis appears to be linked to the interplay of normal skin microbiota (especially *Malassezia* spp.) and thus ensuing impaired immune reaction, the composition of lipids on skin surface and individual susceptibility affected by genetic factors. Additional factors such as drugs, cold temperatures, and stress may exacerbate the condition.

Some risk factors for development of seborrheic dermatitis

- Age
- Male gender
- Increased sebaceous gland activity
- Immunodeficiency
- Neurological and psychiatric disease
- Drugs like dopamine antagonists, immunosuppressants
- Low ambient humidity
- Low ambient temperature

Clinical presentation

The most important clinical feature of seborrheic dermatitis is the characteristic distribution of lesions, which occur in areas rich in sebaceous glands, especially on the scalp and face.

It characteristically demonstrates folliculocentric salmon-colored papules and plaques with a fine white scale, and a yellowish crust often described as a greasy scale-crust. Lesions may present in one or more locations, with less scaling on flexural surfaces. The mildest form of seborrheic dermatitis is a non-inflammatory variant commonly referred to as pityriasis capitis or sicca that affects the scalp and “beard region” and is associated with shedding of small light-colored flakes of skin, often seen as “dandruff” on a background of dark clothing.

Infants

In infants, the disease is generally mild and asymptomatic, but atopic dermatitis can coexist frequently. It usually appears in the 2nd week of life and tends to last 4 to 6 months.

The disease can present in the facial distribution like the adult form, the diaper region, the skin creases of neck, and the axillae. Rash is usually not itchy or painful.

“Cradle cap” is a common presentation, which refers to an adherent yellowish scale-crust that arises on the crown and front of the scalp, developing from a bran-like scale, serous ooze, and a greasy crust, to create a firm mass, which may progress and involve the whole scalp.

Seborrheic dermatitis on head in a child



Adults

Pruritus is common in the adult form of seborrheic dermatitis, especially with scalp lesions. The face, scalp, and chest are the sites that are most commonly involved in adults. On head and neck, it is characteristically symmetrical and involves the central 3rd of the face, the center of the forehead, the eyebrows, the postauricular area, and the external ear canal. The disease characteristically affects the nasolabial folds, and blepharitis is a common finding.

Some common presentations of Seborrheic dermatitis in adults

A. Seborrheic dermatitis in adult face



B. Seborrheic dermatitis in beard area



C. Seborrheic dermatitis at scalp of an adult female patient (back view)



Diagnosis

Seborrheic dermatitis is generally a clinical diagnosis based on the location and appearance of lesions. Investigations are not routinely required in most cases, except in those with sudden-onset severe disease, where HIV serology should be sought. Some other tests that may be helpful in establishing or ruling out the etiology include KOH examination of skin scrapings, swab for microscopy, culture and sensitivities, and histology and direct immunofluorescence.

Treatment

The approach to treatment of seborrheic dermatitis varies according to patient's age and the distribution and severity of condition. Regardless, it is important to educate patients on good general skincare practices, including the use of a soap-substitute and appropriate moisturizing.

Treatments should address not only the underlying disease process, but any secondary features also, like the hyperkeratotic scale, bacterial infection, and associated symptoms, especially pruritus. In infants, gently removing the scale-crust of cradle cap and alleviating parental anxiety appears important. Likewise, in adults, relieving the itch and discomfort seems important.

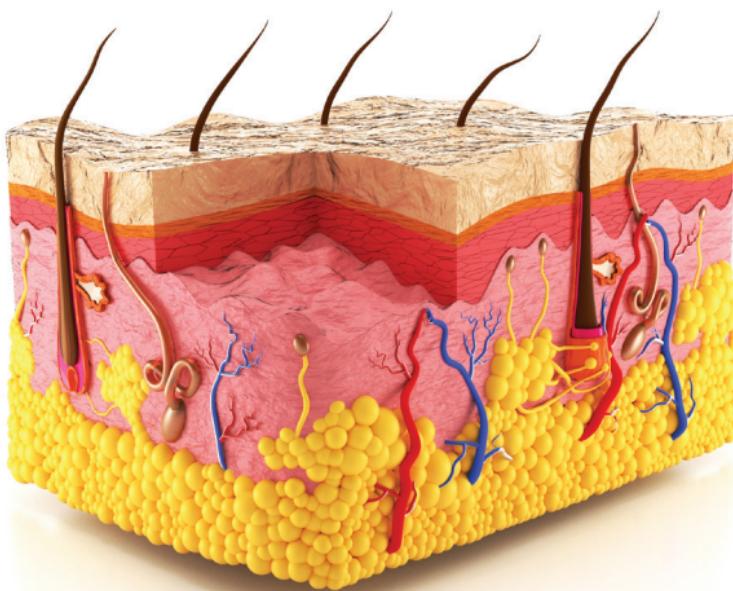
A typical formulary for seborrheic dermatitis could include antifungals, keratolytics, antipruritics, and anti-inflammatories. Often, treatment with an antifungal such as topical ketoconazole is the mainstay of therapy for seborrheic dermatitis of face and body. Anti-inflammatory agents such as topical corticosteroids and calcineurin inhibitors should be used only for short durations given the risk of possible adverse effects.

Several over-the-counter shampoos are also available for treatment of seborrheic dermatitis of scalp, and patients may initiate therapy with one of these agents.

Typical formulary for seborrheic dermatitis may include		
Topical agents (creams, ointments, lotions)	Shampoos	Oral medication
<ul style="list-style-type: none"> • Salicylic acid (2%) + sulfur (2%) in sorbolene cream or emulsifying ointment • Ketoconazole (2%) cream • Clotrimazole (1%) + hydrocortisone (1%) cream • Sulfacetamide (10%) + sulfur (5%) lotion • Betamethasone dipropionate (0.05%) lotion • Tacrolimus (0.03% and 0.1%) ointment 	<ul style="list-style-type: none"> • Zinc pyrithione (1%) • Selenium sulfide (1% to 0.5%) • Ketoconazole (2%) • Ciclopirox (1%) • Coal tar (5%) + salicylic acid (2%) • Tacrolimus (0.1% and 0.03%) 	<ul style="list-style-type: none"> • Itraconazole • Fluconazole • Terbinafine

SECTION 11

DISORDERS OF SEBACEOUS & SWEAT GLANDS



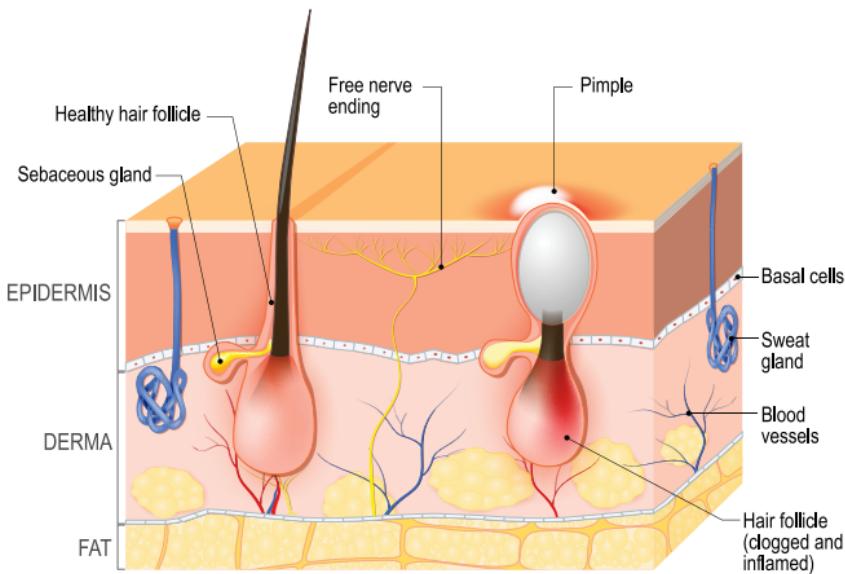
■ DISORDERS OF SEBACEOUS AND SWEAT GLANDS

Disorders of sebaceous and sweat glands are frequently observed in childhood, but may affect any age group. Acne vulgaris is by far the most common disorder, with its peak incidence in adolescence; whereas rosacea affects the face of adults, usually women.

■ ACNE VULGARIS

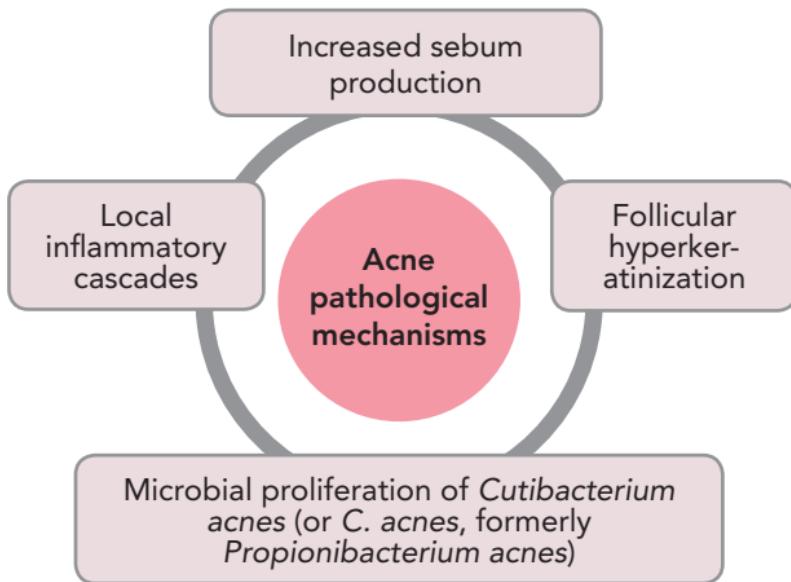
Acne vulgaris is a common skin disorder of the pilosebaceous unit, which although not life-threatening, can have a significant adverse psychological and physical impact.

Acne

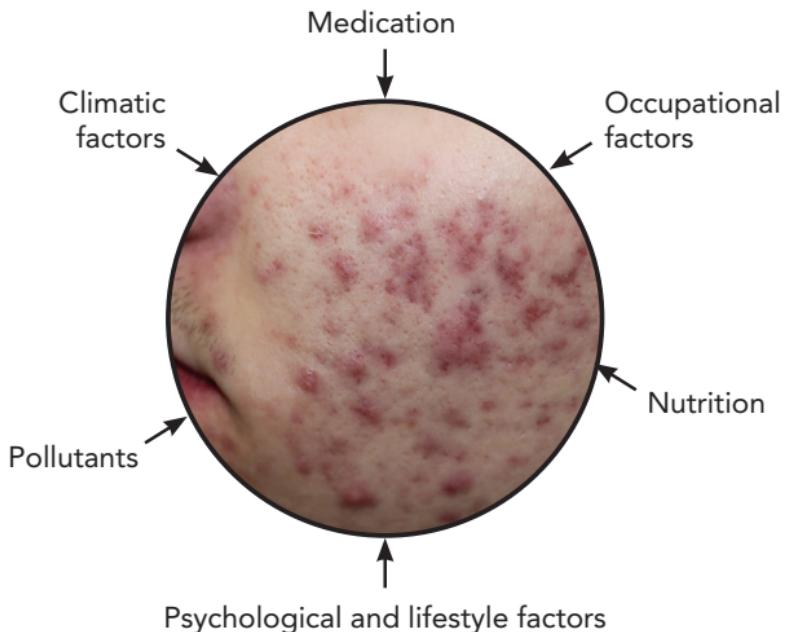


The pathogenesis of acne is primarily inflammatory in nature, but involves a complex interplay of several synergistic, biological mechanisms. The disease occurs by hypersensitivity of the sebaceous glands to circulating androgens, and can be triggered by one or more external factors like nutrition, medications, occupation, pollutants, climate, and psychosocial factors like stress.

Biological mechanisms involved in acne vulgaris



External factors triggering acne



Clinical presentation

Acne vulgaris presents with both inflammatory and non-inflammatory lesions mainly on the face, but can be present on other body parts like upper arms, trunk, and back also.

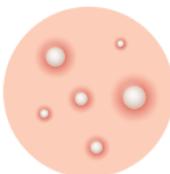
It presents as polymorphic lesions starting with comedones, and can eventually leave various scars after healing.

Acne grading	
Grade 1	<ul style="list-style-type: none">Comedones<i>Open comedones</i> - due to plugging of pilosebaceous orifice by sebum on skin surface<i>Closed comedones</i> - due to keratin and sebum plugging the pilosebaceous orifice below skin surface.
Grade 2	<ul style="list-style-type: none">Inflammatory lesions presenting as small papule with erythema
Grade 3	<ul style="list-style-type: none">Pustules
Grade 4	<ul style="list-style-type: none">Many pustules coalescing to form nodules and cysts

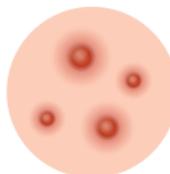
Acne types



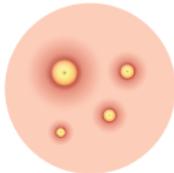
BLACKHEAD



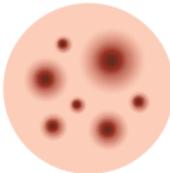
WHITEHEAD



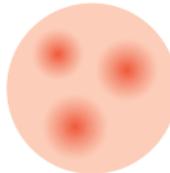
PAPULE



PUSTULE



NODULE



SCAR

Diagnosis

Diagnosis of acne vulgaris is primarily clinical. A history of hirsutism or dysmenorrhea should, however, be sought in women of childbearing age; and if positive, then hormonal levels (testosterone, LH, FSH, and DHEA) should be tested.

Acne vulgaris in a young male



Acne vulgaris and scars on face of a woman



Treatment

There are several therapeutic options available for acne, including both pharmacological (topical and systemic) and non-pharmacological interventions (e.g., lasers and light devices including LED device, photodynamic therapy, intense pulsed light), which can be used alone or in combination in individual patients, to target one or more of the pathogenic processes.

Generally, topical treatment (e.g., benzoyl peroxide, antibiotics, and retinoids) is used as first-line treatment in cases of mild-to-moderate acne with comedonal lesions and inflammatory lesions. Systemic treatment (e.g., oral antibiotics like doxycycline and minocycline) can be used as first-line treatment in cases of moderate-to-severe acne, together with a topical agent.

Treatment options for acne vulgaris in adolescents and young adults		
Mild	Moderate	Severe
First- line treatment		
Benzoyl peroxide (BP) or topical retinoid OR Topical combination therapy (BP + antibiotic, or retinoid + BP, or retinoid + BP + antibiotic)	Topical combination therapy (BP + antibiotic, or retinoid + BP + antibiotic) OR Oral antibiotic + topical retinoid + BP OR Oral antibiotic + topical retinoid + BP + topical antibiotic	Oral antibiotic + topical combination therapy (BP + antibiotic, or retinoid + BP, or retinoid + BP + antibiotic) OR Oral isotretinoin
Alternative Treatment		
Add topical retinoid or BP (if not added already) OR Consider alternate retinoid OR Consider topical dapsone	Consider alternate combination therapy OR Consider change in oral antibiotic OR Add combined oral contraceptive or oral spironolactone (females) OR Consider oral isotretinoin	Consider change in oral antibiotic OR Add combined oral contraceptive or oral spironolactone (females) OR Consider oral isotretinoin

■ ROSACEA

Rosacea is a chronic, multisymptom, inflammatory condition that affects the centrofacial skin, and is frequently associated with facial erythema, which is often troublesome and hard to treat. Although usually limited to skin, an association of rosacea with systemic comorbidities, such as neurologic diseases, inflammatory bowel disease, celiac disease, and cardiovascular diseases has been reported.

Etiology

The exact etiology of rosacea is not fully understood, and could possibly be influenced by several factors, including genetics, activation of the immune system, microorganisms' infestation, environmental factors like ultraviolet (UV) exposure, and neurovascular dysregulation.

Clinical presentation

Rosacea can present with recurrent flushing, erythema, telangiectasia, papules, or pustules on nose, chin, cheeks, and forehead. Secondary manifestations, such as itching, burning, or stinging, are frequently observed.

There are 4 clinical subtypes of rosacea based on the predominant signs and symptoms:

- Erythematotelangiectatic
- Papulopustular
- Phymatous, and
- Ocular.

These subtypes are however not mutually exclusive; and a patient can present with features of more than one subtypes, with predominant features and areas of involvement changing over time. In a large number

of cases, ocular involvement may be there, with symptoms including dryness, redness, tearing, tingling/burning sensation, foreign-body sensation, light sensitivity, and blurred vision.

Clinical subtypes during the disease (Rosacea)	
Subtype	Features
Erythematotelangiectatic	Persistent erythema with intermittent flushing of nose and cheeks; usually the first clinical manifestation of rosacea
Papulopustular	Eruptions of papules and pustules on the affected area on face; sometimes called "adult acne" due to similarity in appearance of lesions. However, a lack of comedones in rosacea helps to differentiate it from true acne.
Phymatous	Fibrosis and hypertrophy of sebaceous glands; typically seen on nose of male patients (rhinophyma), but can also affect cheeks, chin, and glabella.
Ocular	Tearing, dry eye, gritty sensation, pruritus, hordeola, and blepharitis

A young woman with rosacea



Diagnosis

The diagnosis of rosacea is mainly clinical, based on skin manifestations considered in keeping with information about potential triggers. Patients with ocular symptoms should undergo ophthalmic evaluation.

Treatment

The first step in rosacea treatment is to identify the possible triggers such as UV light, spices, and alcoholic beverages, and then advise the patient to avoid them. Further, the choice of therapy is dependent on main signs and symptoms in individual patient, with majority of therapies aimed

to reduce inflammation. Topical steroids should however be avoided owing to the associated risk of rebound flaring.

An ophthalmology referral is recommended in case patient shows any ocular involvement. Topical treatment is recommended in pregnant women. Generally, systemic therapies are used for flares not responding to topical therapy alone; though the latter should be continued to maintain remission thereafter.

Universal skin care recommendations for rosacea patients

- pH-balanced skin cleansers (as opposed to soaps)
- Broad-spectrum sunscreen with \geq SPF 30
- Regular use of moisturizers.

Topical treatments for rosacea

Erythema	Inflammatory papules and pustules
Brimonidine tartrate (alpha-2 agonist) 0.33% gel Oxymetazoline hydrochloride (alpha-1 agonist) 1% cream	Ivermectin 1% cream Azelaic acid 15% gel, foam or 20% cream Metronidazole 0.75% and 1% gel or cream

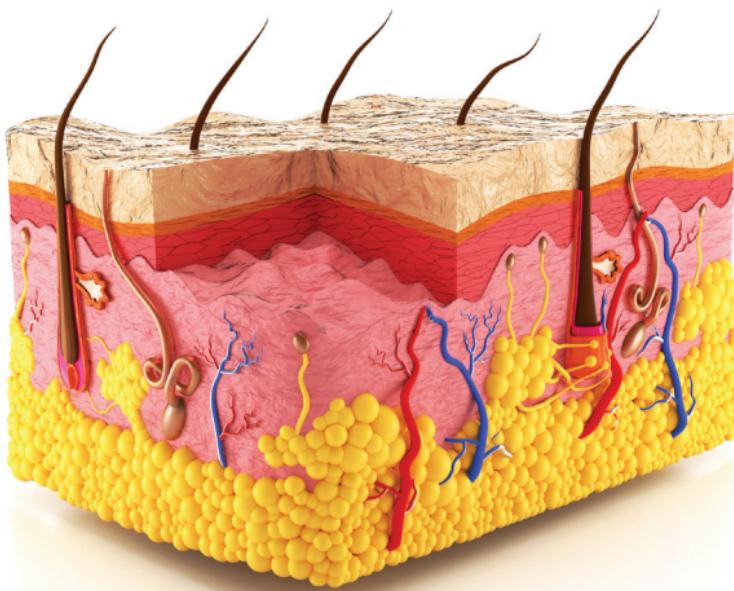
SECTION 11

Systemic treatments for rosacea		
Flushing	Inflammatory papules and pustules	Phyma (inflamed)
Propranolol (20 to 40 mg 2-3 times/day) Carvedilol (6.25mg 2-3 times/day) Clonidine (50 mcg twice daily)	Doxycycline, modified-release (40 mg daily, 30 mg immediate-release and 10 mg delayed-release beads, for 8-12 weeks) Minocycline (50 to 100 mg bd for 8-12 weeks) Tetracycline (250 to 500 mg bd for 8-12 weeks) Azithromycin (250-500 mg 3 times weekly for 4-8 weeks) Isotretinoin (0.25 to 0.3 mg/kg/day for 12-16 weeks)	Doxycycline (100 mg 1 to 2 times daily for 8-12 weeks) Tetracycline (250 to 500 mg bd for 8-12 weeks) Isotretinoin (0.25-0.3 mg/kg/day for 3-4 months)

Procedures/interventions for rosacea	
Erythema/telangiectasia	Phyma (non-inflamed)
<ul style="list-style-type: none">Intense pulsed light therapyNdYAG laserPDL pulsed dye laser 585 to 595 nm	<ul style="list-style-type: none">CO2 laser 10,600 nmSurgical resectionElectrosurgery

SECTION 12

DISORDERS OF PIGMENTATION

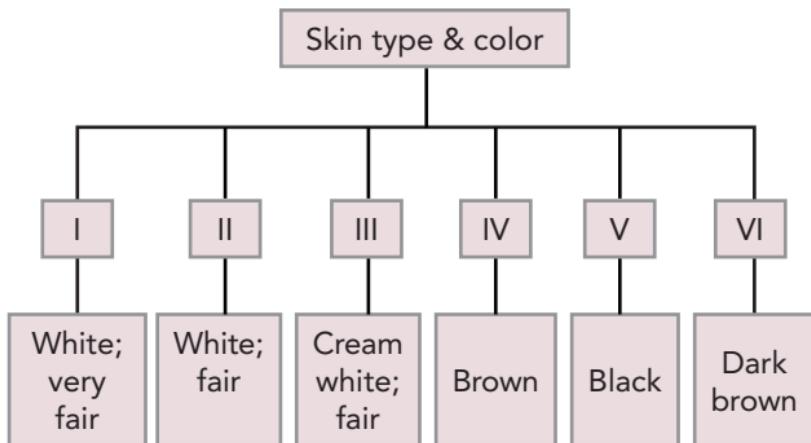


■ DISORDERS OF PIGMENTATION

The skin color is highly individual with a myriad of variations that are controlled genetically. In the skin, the color is decided by the size and the number of the melanosomes. Disorders of pigmentation - hypo- or hyperpigmentation - can have a genetic or acquired cause, and can manifest locally or diffuse.

Certain skin pigmentation disorders are more common in particular skin phenotypes. In India, melasma is the most common pigmentary disorder, followed by the vitiligo. These two disorders are also common among the world's population.

Fitzpatrick classification of skin type/color



■ MELASMA

Melasma is a progressive, macular, non-scaling hypermelanosis of sun-exposed skin that primarily manifests on the face and dorsal forearms, and affects women more than men besides those with darker skin types (IV to VI).

The disorder may be idiopathic, though often associated with pregnancy, or use of oral contraceptives. Although it is usually asymptomatic, it may be cosmetically distressing to the patient.

Etiology

Etiologic factors for melasma include: genetics, UV radiation, pregnancy, hormonal therapies, cosmetics, phototoxic drugs, and antiseizure medications. Melanocytes are stimulated by female sex hormones, producing more melanin pigments when the skin is exposed to sun.

Clinical presentation

Melasma manifests as brown to gray-brown patches on the face, with most people getting it on their cheeks, chin, nose-bridge, forehead, and above the upper lip.

Generally, there are 3 typical patterns of distribution of melasma on the face:

- Centrofacial (mainly)
- Malar, and
- Mandibular

Melasma is usually, but not always, bilateral.

A young women with melasma



Diagnosis

- Epidermal melasma - tends to be light brown, enhancing under Wood Lamp examination
- Dermal melasma – appears grayish in color and is non-enhancing
- Mixed types - dark brown with variable enhancement.

Treatment

- Sunscreens (universally recommended)
- Topical depigmenting therapies (**hydroquinone 4%**, tretinoin 0.05%, and fluocinolone acetonide 0.01%; in triple/dual combination)
- Chemical peels, and light and laser therapies (2nd line)

Treatment is required to be continued to maintain effect. Dermal melasma is generally resistant to topical therapy.

■ VITILIGO

Vitiligo is an acquired immune-mediated hypo-pigmentation disorder, the exact etiology of which remains unknown; though a positive family history can be seen in up to 30% of patients. The disorder can affect all skin types, and is generally considered a cosmetic condition, but can cause significant psychological distress, particularly in those with darker skin types.

Onset of vitiligo is often insidious, but is commonly related to some recent event (stress, illness, or trauma e.g., sunburn); peak onset is in 2nd and 3rd decades.

Clinical presentation

Vitiligo manifests as white macules and patches on the body, caused by an absence of pigmentary cells from the epidermis.

The lesions are characterized by well-demarcated pearly white or depigmented macules and patches, oval, round, or linear-shaped, and the borders are convex, ranging from few millimeters to centimeters and enlarge centrifugally. Koebner phenomenon (development of vitiligo at specific trauma prone sites) is a common clinical manifestation seen in 20% to 60% of patients.

SECTION 12

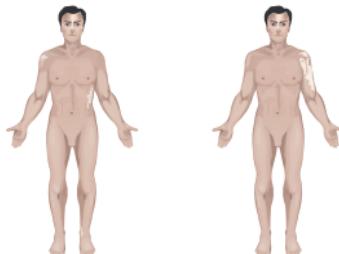
There are two common distribution patterns of vitiligo:

- Segmental - presents as a unilateral/band-shaped distribution of hypopigmented macules, usually 5-50 mm in size and coalescent, with rapid stabilization and early age of onset.
- Non-segmental (i.e., localized and generalized) - typically bilateral or scattered symmetrically and evolves over time; considered generalized when it covers >10% of body surface area.

Acral or acrofacial vitiligo typically involves the face and distal extremities.

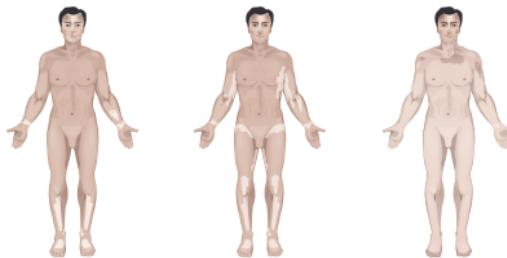
Vitiligo type

LOCALIZED



FOCAL SEGMENTAL

GENERALIZED



ACROFACIAL

VULGARIS

UNIVERSAL

A young girl with vitiligo



Vitiligo on hands



Diagnosis

In most cases of vitiligo, the diagnosis is clear based on clinical findings, though detailed history and examination facilitate assessment of disease severity.

Treatment

There are multiple treatment modalities for vitiligo, often used in combination depending on the distribution; although recurrence is common, as is treatment failure. Generally, lesions of the head and neck tend to be most responsive to treatment, while those present on extremities and genitalia tend to be more recalcitrant.

Sun protection, with use of broad-spectrum sunscreens and sun-protective clothing, is recommended in vitiligo.

Limited vitiligo

- High-potency topical corticosteroids (**betamethasone** 0.1% or fluocinonide 0.05% ointment)
- Topical calcineurin inhibitors (tacrolimus and pimecrolimus)

Generalized or treatment-resistant disease

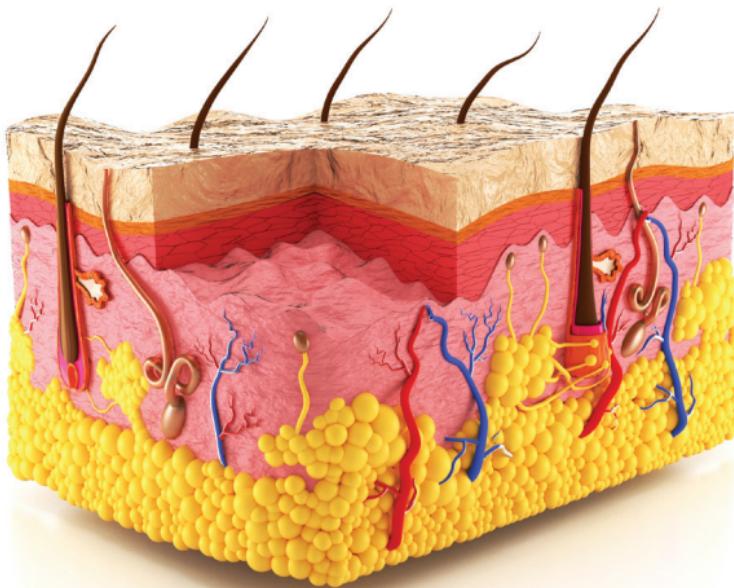
- Systemic therapies (e.g., psoralen and UVA, **narrowband UVB**, systemic corticosteroids)

Extensive generalized vitiligo (>50% of body surface area affected)

- Cryotherapy
- Laser therapy

SECTION 13

PAPULOSQUAMOUS DISEASES

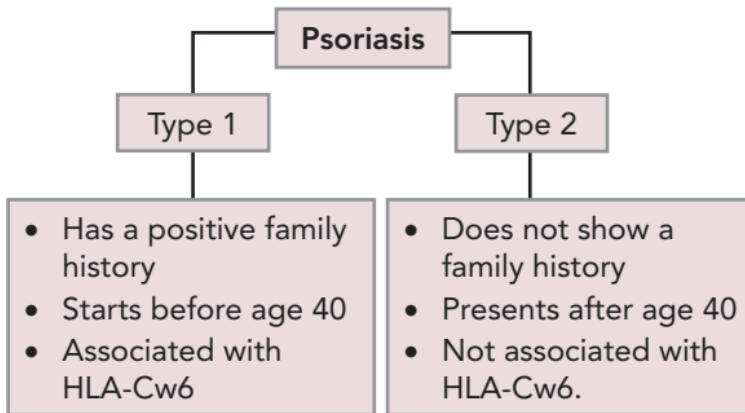


■ PAPULOSQUAMOUS DISEASES

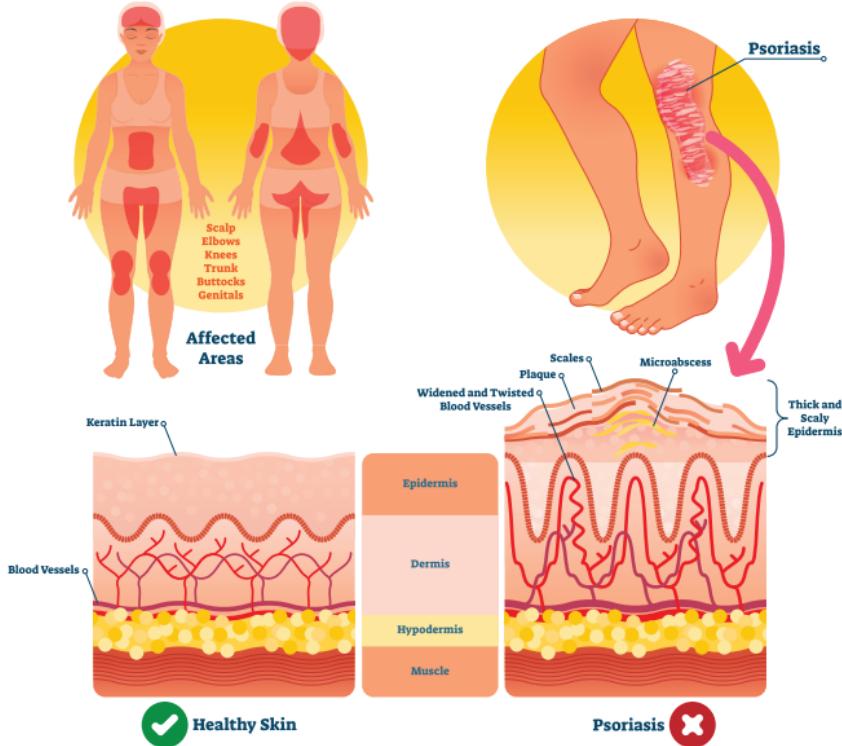
Papulosquamous diseases represent a complex group of disorders characterized by scaly papules and plaques, typically on an erythematous background. These scaly, raised lesions can range in size from small papules (1–3 mm) to large plaques (>10 cm in diameter). Although papulosquamous diseases may have a similar morphology, their underlying etiologies vary. Psoriasis is perhaps the commonest papulosquamous disease observed in clinical practice.

■ PSORIASIS

Psoriasis is a chronic, systemic, immune-mediated, inflammatory disease, caused by a complex interplay of genetic and environmental factors. It is often associated with other autoimmune diseases such as rheumatoid arthritis, Crohn's disease, and thyroid diseases; and can be classified into two types, dependent on family history: type 1 and type 2.



Psoriasis (at a glance)



Etiopathogenesis

The pathogenesis of psoriasis is multifactorial, and the exact etiology remains unclear. Nevertheless, the main cause is considered to be a dysregulation of the cell-mediated, adaptive immune response; likely triggered by hyperactivity of innate immunological system to environmental antigens in genetically predisposed individuals.

This results in hyperproliferation of epidermal keratinocytes, eventually leading to the formation of characteristic psoriatic plaques. The epidermal cells fail to secrete lipids, which result in the typical flaky and scaly skin of psoriasis.

Environmental factors such as stress, low humidity, cold, and disease states such as streptococcal infection, HIV, trauma, medications, and obesity, possibly interact with varying genetic patterns, to precipitate the disease onset and likely accounting for variable expression of the disease. Certain drugs like chloroquine, beta-blockers, steroids, and NSAIDs can worsen psoriasis.

Clinical presentation

Psoriasis patients often show characteristic symmetrical involvement of the extensor surfaces, scalp, and nails.

The most characteristic skin lesion is erythematous plaque covered with silvery scales. Furthermore, approximately, 70-90% of patients with psoriasis also suffer from pruritus; which most often appears at night and in the evening.

Clinically, psoriasis can present with different morphologies in the form of plaque, guttate, erythrodermic, pustular, inverse, and psoriatic arthritis. Variation in site is seen with involvement of scalp, palmoplantar region, genitals, and nails.

Any trauma to the skin in patients with psoriasis induces lesions of psoriasis at that site (**Koebner phenomenon**), indicating the disease activeness.

Different clinical morphologies of psoriasis

1. Plaque psoriasis

- Most common type

- Typically presents as erythematous plaques with silvery scales, most commonly over extensors of extremities (elbows, knees), and scalp, and back.
- **Auspitz sign** - pinpoint bleeding points seen on successive removal of psoriatic scales; confirm the diagnosis clinically.
- Plaques can coalesce and cover large areas of skin.

2. Guttate psoriasis

- Also called as eruptive psoriasis
- Commonly seen in children after an upper respiratory tract infection with streptococci
- Presents with erythematous and scaly raindrop-shaped lesions mainly over trunk and back
- Respond best to phototherapy.

3. Pustular psoriasis

- Presents with small non-infectious pus-filled lesions with erythema surrounding it
- Can be localized or generalized
 - » Localized pustular psoriasis affect the hands and feet
 - » Generalized pustular psoriasis
 - ◊ Associated with hypocalcemia
 - ◊ Presents with sterile pustules on an erythematous plaque involving whole body

4. Erythrodermic psoriasis

- Presents with widespread inflammation in form of erythema and exfoliation of the skin covering >90% of body area

- Associated with severe itching, swelling, and pain
- Results from an exacerbation of unstable plaque psoriasis, following abrupt withdrawal of systemic steroids.

5. *Inverse psoriasis*

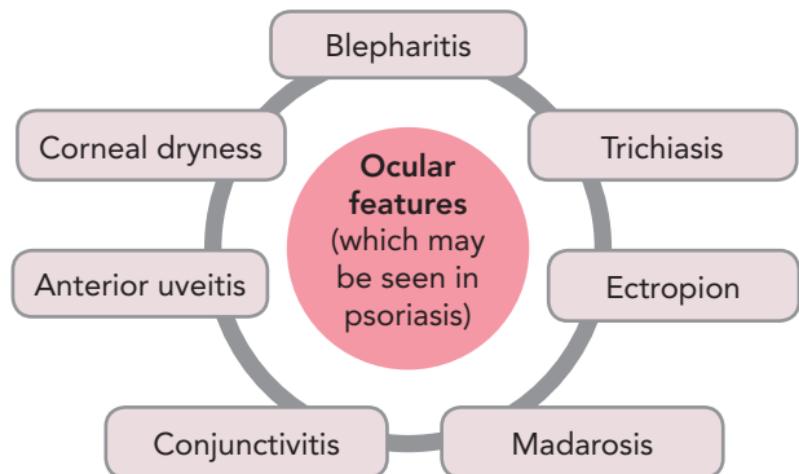
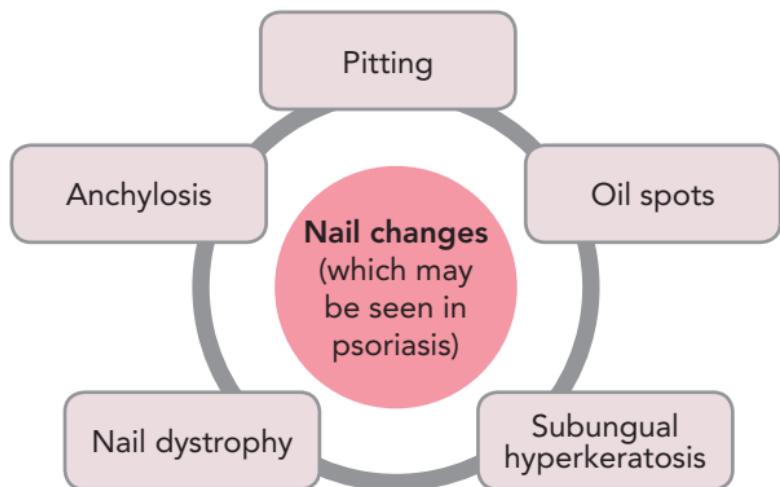
- Also called as flexural psoriasis or intertriginous psoriasis
- Appears as smooth, erythematous and sharply demarcated patches affecting intertriginous areas like groins, armpits, intergluteal region, inframammary region.
- Skin may be moist, macerated, may contain fissures which may be malodorous, pruritic, or both.
- Needs to be differentiated from dermatophyte infection at these sites that present with central clearing and active border with scales, vesicles, and pustules at margin

6. *Sebopsoriasis*

- Typically manifests as red plaques with greasy scales
- Commonly affects areas with increased sebum production, such as scalp, forehead, nasolabial folds, and retro-auricular folds.

7. *Psoriatic arthritis*

- Chronic inflammatory arthritis, commonly occurring in association with skin and nail psoriasis
- Typically involves painful inflammation of joints and connective tissue; commonly affects joints of fingers and toes
- Leads to sausage-shaped swelling of fingers and toes (**dactylitis**)
- Can also affect hips, knees, spine and sacroiliac joints



Fissured tongue is the most common finding of oral psoriasis.

SECTION 13

Some common presentations of Psoriasis in adults:

A. Psoriasis on forehead



B. Psoriasis on back



C. Psoriasis on foot



D. Psoriasis on knee



SECTION 13

E. Psoriasis at the nail



F. Psoriasis on hands



G. Psoriasis on elbows



Diagnosis

The diagnosis of psoriasis is usually clinical, made by the morphology and site of lesions. In cases with uncertainty, histopathology may help to differentiate it from another dermatosis.

Characteristic features on biopsy:

- Parakeratosis
- Micro-abscess
- Absence of granular lesions
- Regular elongation of ridges in form of camel foot appearance
- Spongiform pustules with dilated and tortuous capillaries in dermal papilla

Treatment

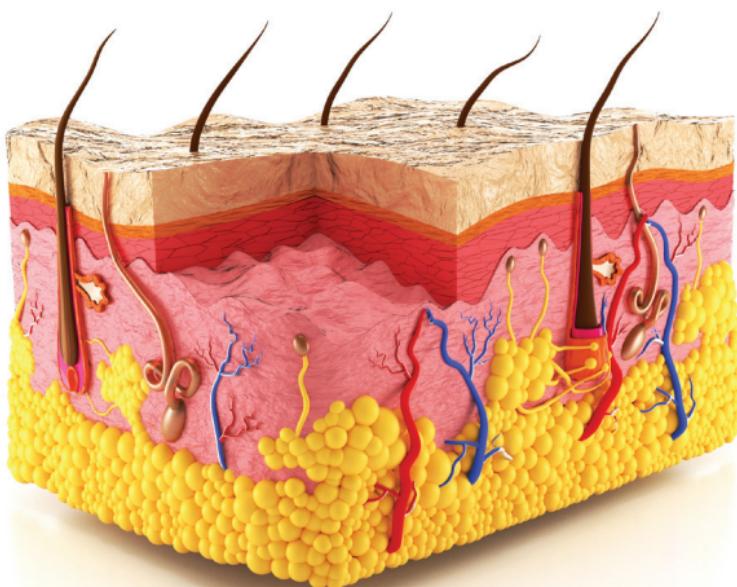
Treatment options for psoriasis include both topical and systemic agents, which are used dependent on the patient profile and disease severity. Psoriasis Area Severity Index (PASI) is a widely used measurement tool to assess the severity and evaluate treatment efficiency.

- In general, topical therapy is used in mild to moderate psoriasis, and systemic therapies (e.g., methotrexate, retinoids, cyclosporine, and fumarates) are used in extensive cases, the involvement of nails and psoriatic arthritis
- Topical agents used include dithranol, corticosteroids, vitamin D analog, and retinoids.
- Methotrexate is the drug of choice that should be used as long as effective. Intermittent cyclosporine can be used to induce a clinical response.
- If there is no response to methotrexate, biological agents (e.g., adalimumab, infliximab, etanercept, certolizumab pego, and interleukin inhibitors) can be used; in some cases, combined with methotrexate. However, patient should be tested for infections (like tuberculosis and hepatitis) before starting any biological agent; and all precautions be taken to avoid severe immunocompromise.

- Phototherapy includes PUVA therapy (psoralen + UVA), as well as narrowband UVB light with a range of 311 to 313 nanometers. Narrowband UVB is equally effective without the side effects of psoralen, and can be given to children, pregnant and lactating females, and elderly.
- Monitor routine blood, liver functions, and renal functions in patients on systemic therapy.
- Aggressive treatment with topical corticosteroids is required for ocular psoriasis.
- Adjunctive measures:
 - » Use emollients and moisturizers to improve barrier function and retain skin hydration.
 - » Avoid skin trauma for fear of inducing the Kobner reaction
 - » Avoid use of beta-blockers, chloroquine or NSAIDs.
 - » Avoid alcohol
 - » Include diets high in fish oils.

ANNEXURE

ZINC IN DERMATOLOGY



■ ROLE OF ZINC IN VARIOUS DERMATOLOGICAL CONDITIONS

In the human body, zinc is associated with a myriad of organic activities, such as development, differentiation, and cell growth; and this has significance with respect to skin also.

In fact, skin is the 3rd most zinc-abundant tissue in the body; where it is present in differential concentration in different layers of the skin, and together with its transporters, contributes in various skin functions and skin homeostasis.

The nutrient plays an important role in maintaining integrity of intercellular structures that promote adhesion between epithelial cells and that are necessary for the epithelial tissue structure.

Some reported functions of Zinc and Zinc transporters in skin cells	
ZIP2 & ZIP4 (<i>in keratinocytes</i>)	Facilitate keratinocyte proliferation and differentiation
ZIP10 (<i>in epidermal progenitor cells in outer root sheath</i>)	Facilitate proper epidermal formation
ZnT5 (<i>in mast cells</i>)	Involved in inflammatory cytokine production
Many ZnTs & ZPs (<i>in dendritic cells</i>)	Regulate MHC class II expression
ZIP8 (<i>in T cells</i>)	Involved in interferon- γ (IFN- γ) production

Some reported functions of Zinc and Zinc transporters in skin cells	
<i>ZIP7 (in fibroblasts)</i>	Required for dermal formation
<i>ZIP13 (in fibroblasts)</i>	Required for bone morphogenetic protein/transforming growth factor- β (BMP/TGF- β) signaling
<i>ZIP13 (in adipocytes)</i>	Inhibits beige fat cell differentiation
<i>ZIP14 (in adipocytes)</i>	Suppresses an excess inflammation

Zinc deficiency can therefore be associated with several skin manifestations, such as dermatitis, alopecia, acne, eczema, dry, and scaling skin; while its supplementation may improve the tissue function.

In patients with steroid responsive dermatoses like eczemas, addition of zinc sulphate to steroid like clobetasol propionate has been shown to improve the efficacy of the topical preparation. Likewise, systemic use of zinc may also have potential applications in several dermatological conditions.

Potential dermatological conditions amenable to zinc therapy

Potential uses of zinc in dermatology

Topical zinc therapy

- Warts
- Herpes genitalis
- Dermatophytoses
- Pityriasis versicolor
- Acne vulgaris
- Psoriasis
- Eczemas
- Ulcers
- Melasma
- Ageing

Systemic (oral) zinc

- Warts
- Acne vulgaris
- Rosacea
- Psoriasis
- Ulcers
- Vitiligo

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* E Nettis et.al : J Asthma Allergy. 2009; 2: 17-23.

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* Church et.al : Current Medical Research and Opinion 2020, Vol. 36, No. 3, 445-454

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* G Faghihi et. Al : J Eur Acad Dermatol Venereol. 2008 May;22(5):531-6.

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