Primary Care Dermatology Care Guide

July 2025

CALIFORNIA CORRECTIONAL
HEALTH CARE SERVICES

Information contained in the Care Guide is not a substitute for a health care professional's clinical judgment. Evaluation and treatment should be tailored to the individual patient and the clinical circumstances. Furthermore, using this information will not guarantee a specific outcome for each patient. Refer to "Disclaimer Regarding Care Guides" for further clarification.
https://cchcs.ca.gov/clinical-resources/

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GOALS

- ✓ **Diagnose patients of all skin tones and hair textures presenting with common dermatologic diagnoses.**
- ✓ **Recognize dermatologic signs of systemic disease.**
- ✓ **Appropriately refer patients to specialty care or higher level of care (HLOC) as clinically indicated**
- ✓ **Counsel patients on maintaining healthy skin, hair, and nails.**

ALERTS

- **Understand the cultural aspects in providing dermatologic care.**
- **Recognize the benefits of coordinated care from primary care to dermatology**
- **Appreciate the potential adverse effects of systemic medications, like oral corticosteroids and therapeutic biologics**

INTRODUCTION

Dermatologic concerns are common complaints in primary care that require early detection and intervention. Several studies have demonstrated that most patients presenting with dermatologic concerns can be managed in primary care; approximately 10% of these patients were referred to dermatology. This care guide aims to support providers with a systematic approach to evaluating patients that present with dermatoses, to distinguish normal from abnormal findings, to integrate pertinent symptoms and signs into an appropriate differential diagnosis, and to enhance awareness of common dermatologic diagnoses encountered in primary care.

HEALTH EQUITY ALERT

Contributing factors that cause significant racial and ethnic disparities in dermatologic care include socioeconomic factors, gaps in medical education, and underrepresentation of minority groups in the dermatology workforce. Furthermore, this underrepresentation of skin of color (SoC) in medical education negatively affects provider confidence in their diagnosis of these patients, thereby compromising timely treatment.[2]

EVALUATION

HISTORY

For any patient presenting with a dermatologic concern, get a detailed history, which should include the following:

Demographic Information

- **Age**: Many conditions have a predilection for certain age groups.
- **Sex and gender**: Both sex and gender have a distinct impact on the incidence, prevalence, clinical presentation, severity, therapeutic response, and associated mental health impacts of dermatologic conditions.
- **Institution**: Inquiring about recent contacts or exposures may be important in infectious disease outbreaks.

Symptom

- **Site and spread**: Ask about the location of the dermatologic concern, which can be localized, patchy, or generalized. Also ask about details of the spread.
- **Duration**: Clarify when the patient first noticed the dermatologic concern, which can be a new symptom of abrupt or gradual onset, a chronic but stable condition, or a relapse of a chronic condition.
- **Timing**: Determine if there is fluctuation or persistence of the dermatologic concern, which may improve on days when a patient is not exposed to sunlight or occurs only during hot weather.
- **Associated symptoms**: Ask about specific characteristics of the dermatologic concern, such as itching, burning, soreness, pain, weeping, oozing, blistering, bleeding, or foul odor
- **Provoking or aggravating factors**: Some infectious or drug triggers may precede a complaint by several days or weeks.
- **Previous treatments**: Discuss the response or lack of response to prior treatments

Evaluation, cont'd

Personal: Smoking, poor dietary habits, personal hygiene

- Associated **medical conditions**: impaired glucose tolerance, diabetes, obesity, cirrhosis, hepatitis, HIV, cancer, chronic kidney disease, asthma, allergies, or autoimmune/inflammatory disease (e.g., lupus, psoriatic arthritis).
- Inquire about **sexual health history** for suspected sexually transmitted infections (STI)
- Evidence supports the association between chronic dermatologic conditions and **mental health** issues, such as depression, anxiety and suicide risk. Care coordination and collaboration with mental health is very important.
- **Oral health** and mucocutaneous inflammation affect many dermatologic conditions, especially those that also involve the oral mucosa. Aphthous stomatitis is correlated with poor periodontal health and greater plaque accumulation. Atopic dermatitis shows an association with gingivitis, toothaches, and oral infections. Heavier enamel plaque burden and reduced oral care are implicated in the exacerbation of lichen planus. Mucous membrane pemphigoid and pemphigus are intimately influenced by oral health, underscoring the important role of good oral health and hygiene. Psoriasis presents a strong connection with oral streptococcal bacterial burden, has been shown to be improved or even cured with tonsillectomy, and has treatment outcomes that are generally associated with periodontal disease. Consider referral to dental to comanage patients.

Medications: Topical and systemic medications, both prescribed and over-the-counter products may result in adverse effects of the skin, hair, and nails

Family History: Consider psoriasis, atopic dermatitis, hidradenitis suppurativa, melanoma[1]

PHYSICAL EXAM

STEP 1: PREPARATION
Perform a dermatologic examination in a well-lit room. You may need a disposable paper ruler, a handheld source of light with magnification, hand sanitizing gel or lubricating jelly, and a glass slide. Announce which body part you will be examining and ask permission from the patient to fully uncover the area. The patient may need to be in a gown for adequate inspection of the body part.

STEP 2: INSPECTION
When inspecting the scalp, include evaluation of the hair follicles and the hair shaft to the tips. Oral mucosal findings may aid in diagnosis. When inspecting the hands and feet, include the web spaces. Magnify the area if needed to determine the texture of smaller lesions and the surrounding skin.

STEP 3: PALPATION
Palpate lesions:

- Determine if lesion are flat or raised
- Assess consistency, texture, and depth of lesion(s)/rash
- Evaluation for tenderness

STEP 4: SIZE
Use the disposable paper ruler to:

- Measure solitary skin lesions in 2 or 3 dimensions, as clinically indicated
- Record measurements in EHRS
- If patient consents to clinical photography, document patient name, CDCR #, area of skin being photographed, and date of examination on paper ruler, and include the ruler for measurement in the clinical photograph
- Follow lesion progression over time, if clinically indicated.

HEALTH EQUITY ALERT

The dermatologic examination often requires assessment of sensitive anatomy, including breasts, buttocks, and genitalia. This can be emotionally invasive and risk the therapeutic alliance if not performed in accordance with trauma-informed care.
Key components of trauma-informed care for dermatologic exams include:

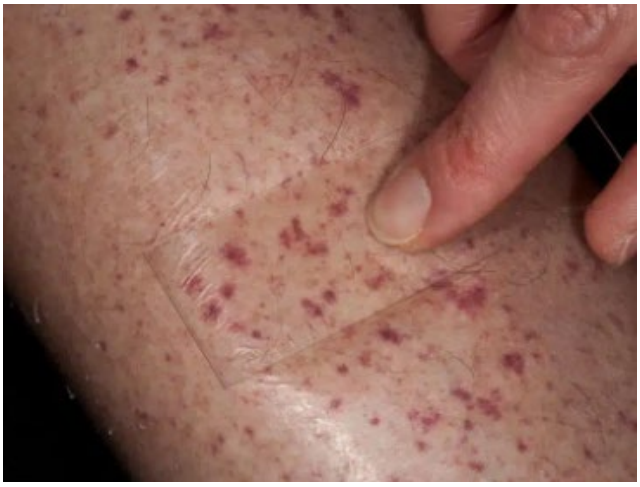
- Age, sex assigned at birth, gender identity, culture, and language
- Clear and direct communication that validates their emotions and hesitancy, describes the steps of the dermatologic examination, explains the reason for the examination, and provides options and choice
- Explicit informed consent prior to the patient disrobing
- Performing patient-perceived invasive exams and sensitive exams once and in the presence of a chaperone
- For lesions suspicious for trauma, gentle investigation for further discussion
- Time – allow the patient the time to discuss their hesitancy, the time to understand the indication, benefits, risks, and alternatives to disrobing for the dermatologic examination, and the time to proceed with the examination at their own pace, if clinically permissible
- Patient agency to withdraw consent and pause or terminate the dermatologic examination

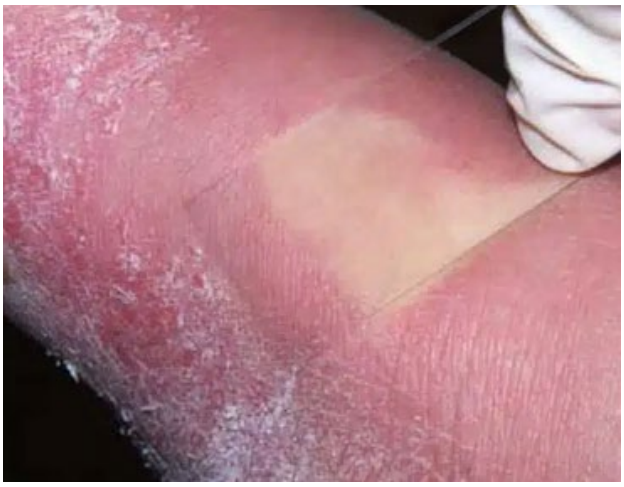
Pursue culturally and linguistically competent methods that prioritize their physical and emotional safety.

Evaluation, cont'd

STEP 5: DIASCOPY

Use the glass slide to press down on the skin, if clinically indicated. Diascopy helps differentiate between blanching rashes and non-blanching ecchymoses, petechiae, and purpura





STEP 6: SCALE AND OTHER SECONDARY SKIN CHANGES

Assess the **primary lesion** and surrounding skin for **secondary lesion(s)**. **Primary lesions** are those that arise de novo. **Secondary lesions** are those are those that occur through the natural evolution of a disease process and by the manipulation of primary lesion. For example, a patient with a contact dermatitis (primary lesion) from their wristband may present with excoriations (secondary lesion) from scratching.

Using a glass slide:

- Rub the edge of the glass slide to gently scrap the lesion to determine if subtle scale is present
- Scrape any scale onto the glass slide to examine microscopically for fungal infection, if clinically indicated

STEP 7: MORPHOLOGIC DESCRIPTIONS

Before a diagnosis can be determined, one of the most important aspects of a dermatologic exam is the accurate description and classification of skin lesion morphology with detailed descriptors.

Primary Morphology: Skin lesions can be flat, raised above the level of the surrounding skin, or depressed below the plane of the skin. Morphology describes the form or structure of a dermatosis. Other morphology descriptors may include:

- Dermatoses can be cooler or hotter than the surrounding skin.
- Skin lesions can be mobile or immobile.
- Consistency of a skin lesion can be described as soft, firm, hard, fluctuant, or sclerosed/scarred.
- The surface of a skin lesion may be normal or smooth if the pathologic process is dermal or subcutaneous. Surface changes indicate epidermal changes.
- If multiple lesions are present, describe their configuration to each other as scattered, clustered, grouped, linear, zosteriform, coalescing

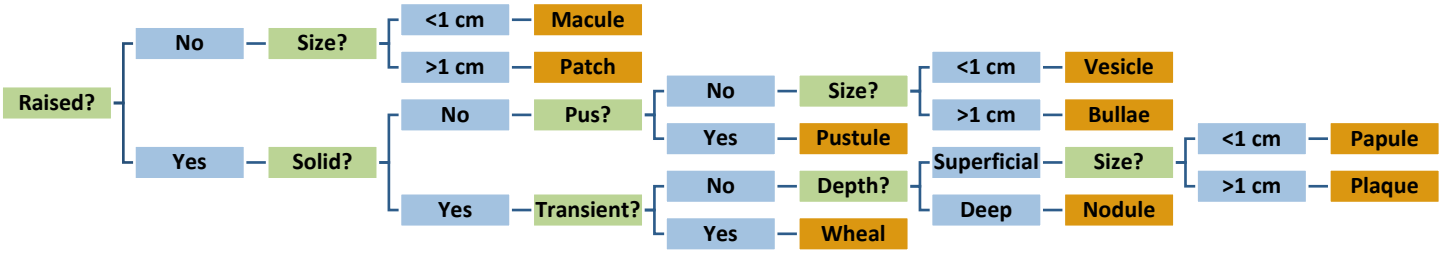
Configuration: Describe the shape of the lesions
Demarcation: Describe the border as clearly defined or not defined.
Color: Skin lesions can be skin-colored, red, pink, violaceous, or any other color. Rashes can be blanching or non-blanching ecchymoses, petechiae, and purpura.
Distribution: Dermatoses and rashes can be scattered or spread over region or in a pattern.
Secondary Morphology: Determine skin characteristics and morphologies that evolved from the primary lesion, traumatic injury, or other external factors.[1]

Evaluation, cont'd

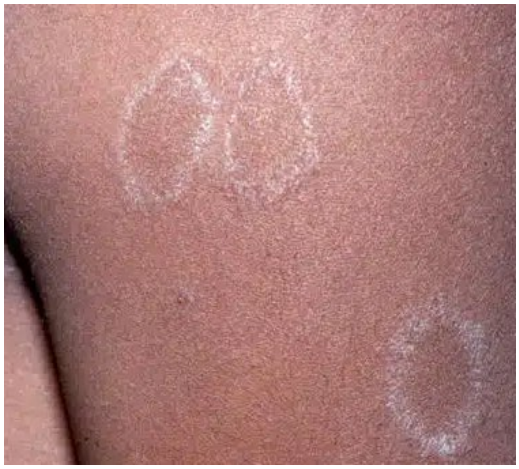
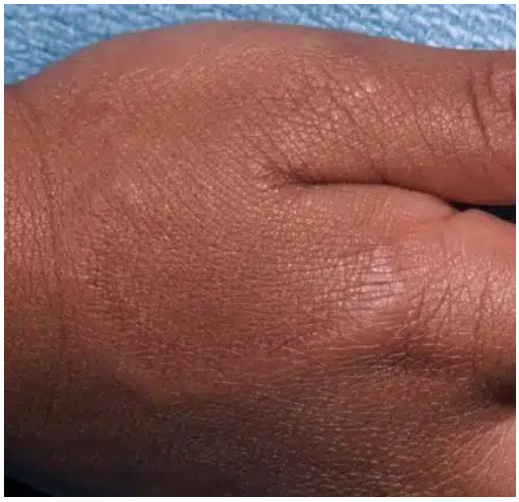
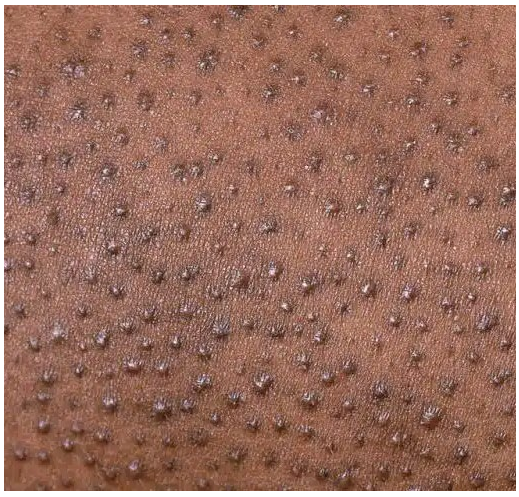
Morphology of Primary Lesions				
Terminology	**Diameter**	**Description**	**Picture**	**Clinical Examples**
Macule	< 1 cm	Level with skin surface, differs in color from bordering skin		Lentigo, petechiae
Patch	> 1 cm	Large area of nonpalpable skin with a color change		Vitiligo, tinea versicolor
Papule	< 1 cm	Solid, elevated lesion		Angioma, wart
Plaque	> 1 cm	Circumscribed, palpable, elevated lesion		Psoriasis, lichen planus
Nodule	> 1 cm	Dermal or subcutaneous solid elevated lesion		Amelanotic melanoma
Wheal	Any size	Edematous, smooth papule or plaque that lasts < 24 hours		Urticaria (hives), dermatographia
Vesicle	< 1 cm	Clear, fluid filled lesion		Herpes, mpox

Evaluation, cont'd

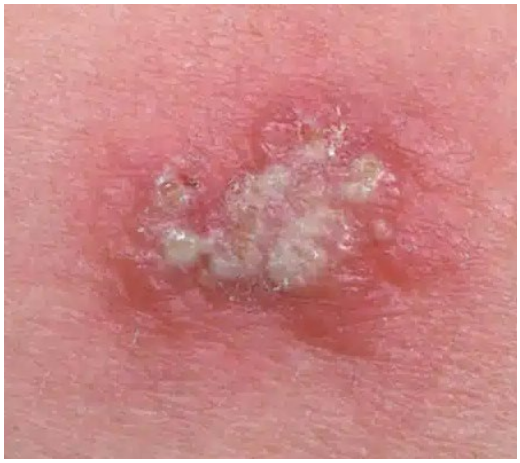
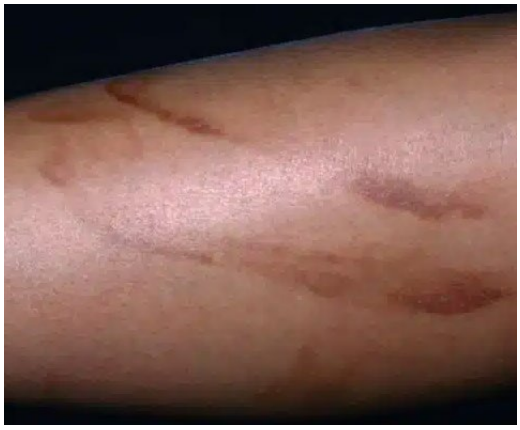
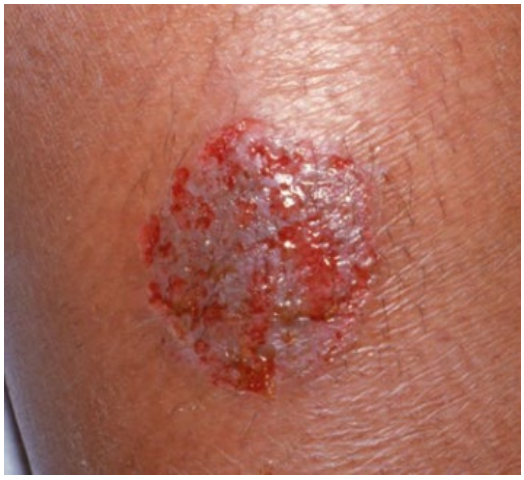
Morphology of Primary Lesions				
Terminology	**Diameter**	**Description**	**Picture**	**Clinical Examples**
Bulla	> 1 cm	Circumscribed lesion containing fluid or blood		Bullous pemphigoid
Pustule	< 1 cm	Cavity filled with pus, may be sterile		Acne, rosacea, folliculitis
Cyst	> 1 cm	Cavity filled with fluid or semi-fluid material		Epidermal inclusion cyst
Abscess	Any size	Localized collection of pus		Skin and soft tissue infection
Tumor	Any size	Solid mass or neoplasm, which can be benign or malignant		Nevus, basal cell carcinoma



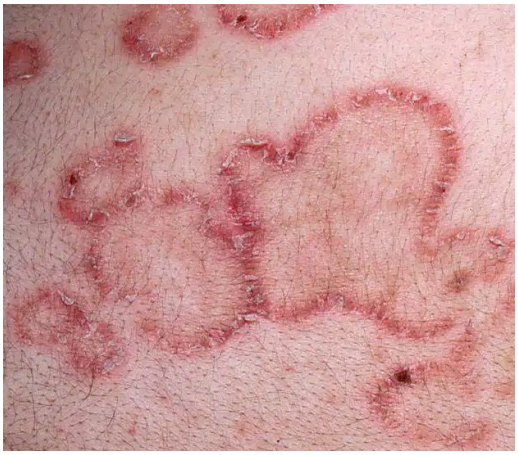
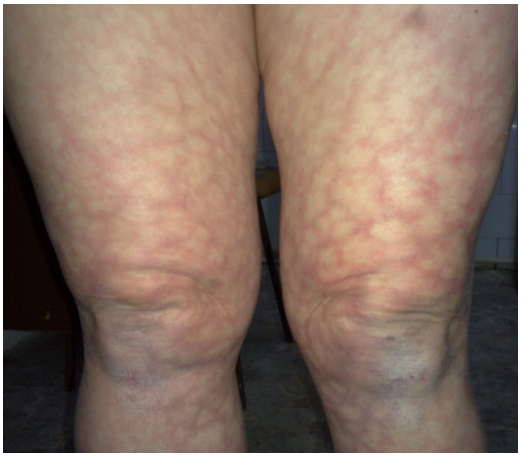
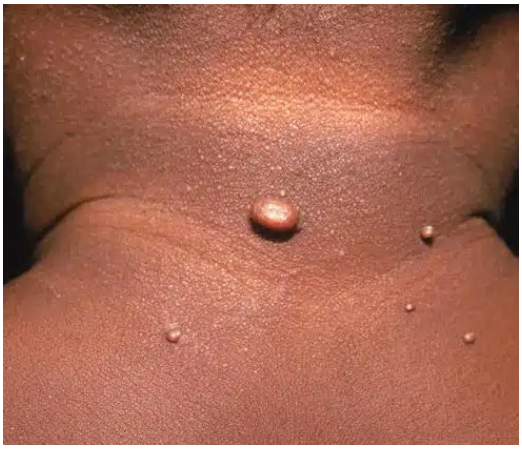
Evaluation, cont'd

Description of Lesion Configuration		
Terminology	**Description**	**Picture**
Annular	Ring-shaped	 Pityriasis rosea
Arcuate	Arc-shaped	 Granuloma annulare
Follicular	Related to hair follicles	 Keratosis pilaris

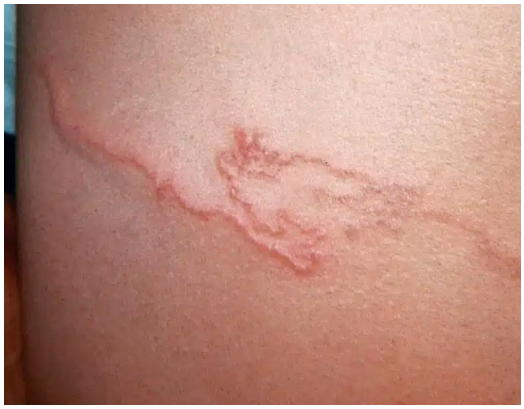
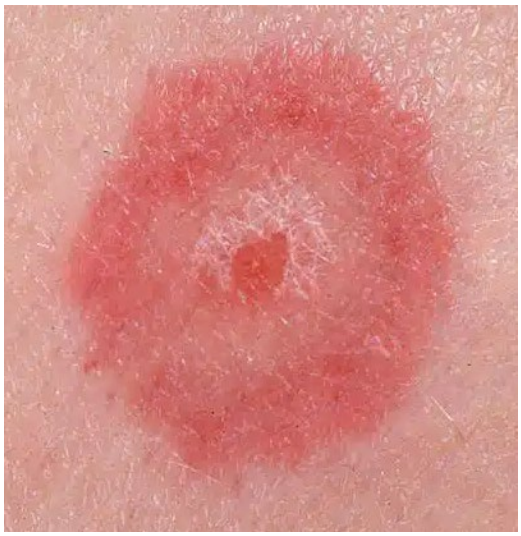
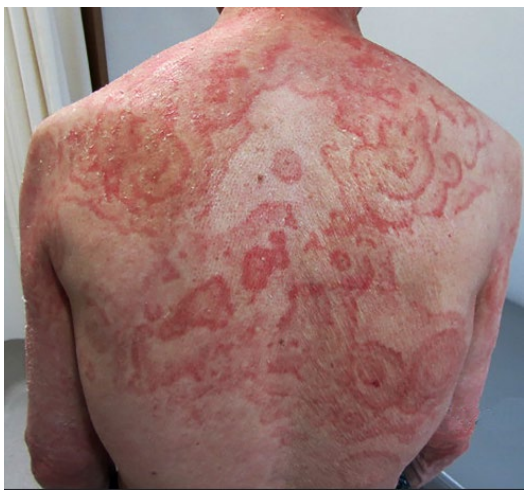
Evaluation, cont'd

Description of Lesion Configuration		
Terminology	**Description**	**Picture**
Grouped	Clustered	 Herpes simplex virus lesion
Linear	Resembling a straight line	 Drug-induced flagellate pigmentation
Nummular	Circular or coin-shaped	 Nummular eczema

Evaluation, cont'd

Description of Lesion Configuration		
Terminology	**Description**	**Picture**
Polycyclic	Coalescing circles or rings	 Necrolytic migratory erythema (glucagonoma syndrome)
Reticular	Netlike	 Livedo reticularis
Scattered	Irregular distribution	 Molluscum contagiosum

Evaluation, cont'd

Description of Lesion Configuration		
Terminology	**Description**	**Picture**
Serpiginous	Serpentine or creeping	 Cutaneous larva migrans
Targetoid	Concentric circles that look like a bullseye	 Erythema multiforme
Whorled	Spiraled appearance	 Erythema gyratum repens (paraneoplastic)

Evaluation, cont'd

Description of Skin Surface	
Terminology	**Description**
Scaling	Increase in the dead cells on the surface of the skin (stratum corneum) causing flaking
Desquamation	Skin peels off in sheets or scales
Eczematous	Pruritic, inflammatory papular/papulovesicular eruption
Psoriasiform	Large white or silver flakes
Pityriasiform	Branny powdery scale
Lichenoid	Scale tightly adherent to skin surface
Keratotic	Horny scale
Hyperkeratotic	Excessive buildup of keratin causing thickening
Exfoliation	Peeling skin
Erythema	Classically described as bright red; however, can appear as violaceous, ashen grey, or darker brown in dark skin tones
Erythroderma	Also called exfoliative dermatitis; severe, life-threatening clinical sign that presents as diffuse, confluent erythema and scaling involving ≥ 90% of the total body surface area
Maceration	Moist peeling skin
Verrucous	Warty skin
Morbilliform	Resembling measles, depicted by erythematous macular or maculopapular rash
Petechiae	Non-blanching red-purple macules
Purpura	Hemorrhagic lesions larger than petechiae and result from bleeding within skin or mucous membranes from capillaries and other small blood vessels
Telangiectasia	Dilated superficial blood vessels in the outer layer of the skin
Ecchymosis	Extravasation of blood into the skin or mucous membranes
Atrophy	Thinning of tissue defined by its location, such as epidermal atrophy, dermal atrophy, or subcutaneous atrophy
Eschar	A necrotic scab or dry crust that results from trauma, infection, or excoriating skin disease
Gangrene	Necrotic, usually black, tissue due to obstruction, diminution, or loss of blood supply, which may be described as wet or dry: • Wet gangrene often follows a crushing injury, has an offensive odor, and spreads rapidly • Dry gangrene becomes cold, dry, and shriveled and eventually turns black

Evaluation, cont'd

Description of Distribution	
Terminology	**Description**
Isolated	Solitary of single
Localized	Limited to or discreet area of skin
Generalized	Randomly distributed lesions over many body regions or without an anatomical region; also called scattered
Disseminated	Dispersed or spread widely throughout the whole body; also called widespread
Acral	Affecting the distal extremities, such as ears, fingers, toes, nose, penis, and nipples
Langer lines	Also known as cleavage lines, following orientation of collagen fibers in the dermis; generally parallel to underlying muscle fibers (See Figure 1)
Dermatomal	Following segmental pattern along skin innervated by cutaneous branches of a single spinal nerve (See Figure 1)
Blaschkoid	Following segmental pattern along pathways of epidermal cell migration and proliferation during embryogenesis; generally, longitudinally oriented on limbs and circumferentially on trunk (See Figure 1)
Lymphangitic	Lying along the distribution of lymph vessel
Photodistributed	Affecting areas of skin that receive the greatest exposure to sunlight
Extensor	Involving skin overlying extensor surfaces of limbs
Flexural	Involving areas of skin folds along flexion surfaces of links
Intertriginous	Involving areas of skin folds where two opposing skin surfaces are in contact (i.e., axilla, inguinal folds, inner thighs, under abdominal pannus)
Inframammary	Intertriginous area of skin folds below breasts
Koebnerize	Involved skin at site of trauma or irritation
Follicular	Individual lesions arising from hair follicles

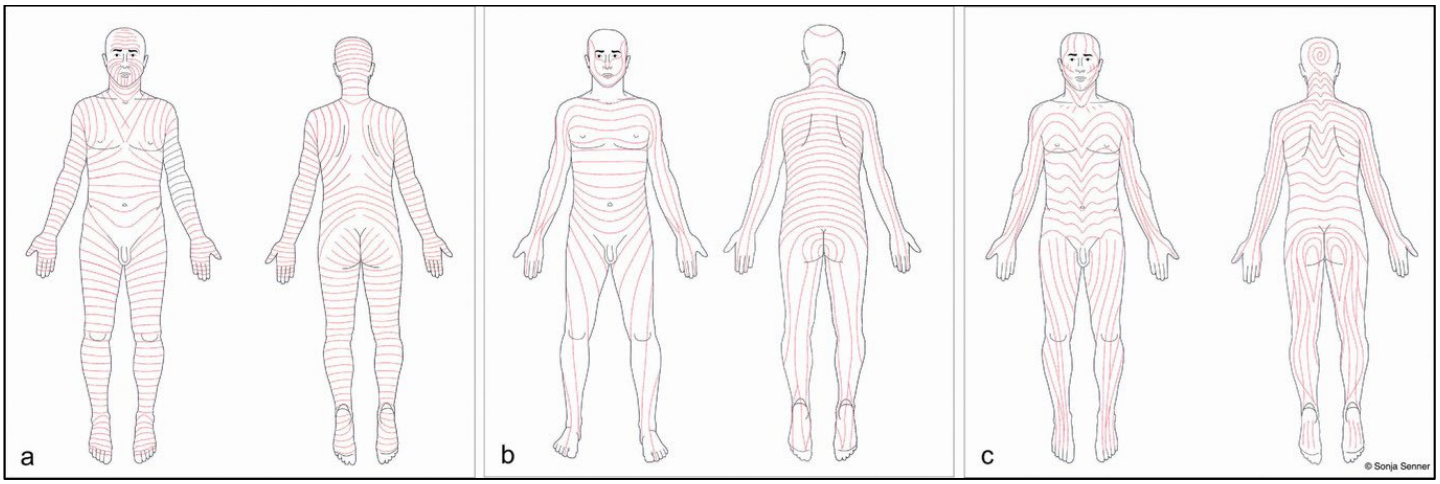
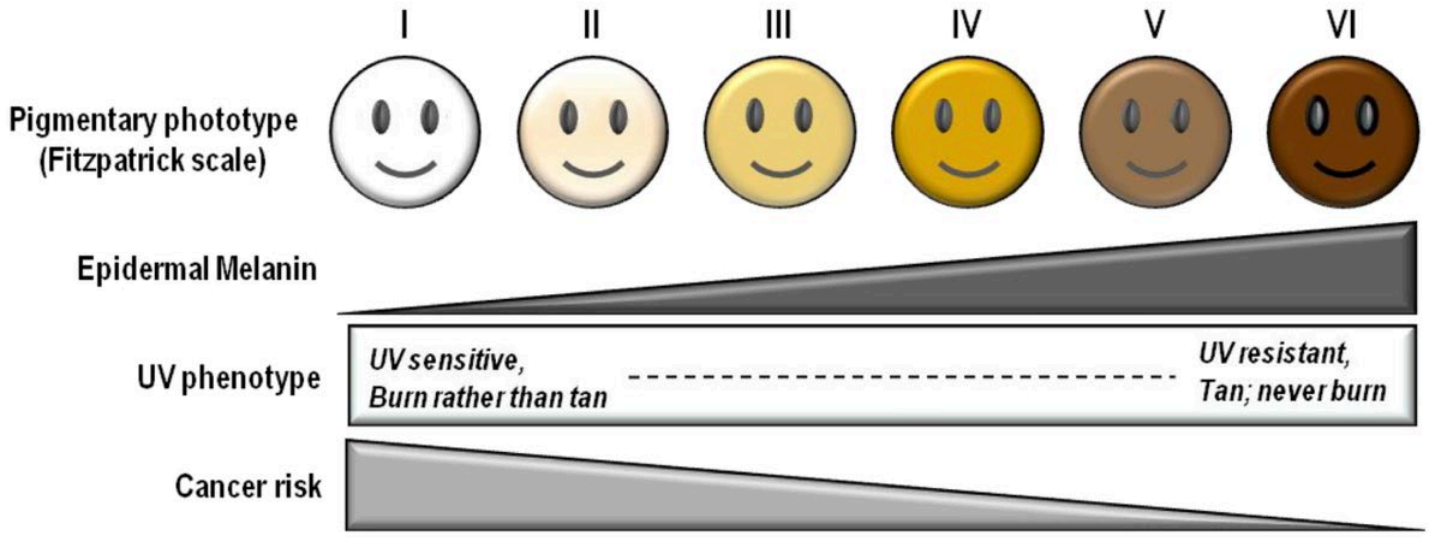


Figure 1: Three clinically relevant linear patterns cover the human body
(a) Langer lines, (b) dermatomes, (c) Blaschko lines

Evaluation, cont'd

Diascopy	
Blanching	**Non-blanching***
Erythroderma	Idiopathy thrombocytopenia
Telangiectasia	Hemolytic uremic syndrome
Sunburn	Vasculitis rashes
Atopic dermatitis and eczema	Acute meningococcemia
Urticaria	Bacterial sepsis
Acne vulgaris	Infective endocarditis
Melasma	Spotted Fever Rickettsioses
Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis	Strongyloidiasis
Erythema Multiforme	Severe dengue with petechiae
Erythema Nodosum	Late viral rash (petechial rash)
Staphylococcus scalded skin syndrome	Forceful coughing, vomiting, excessive crying or straining
Tinea and dermatophytosis	Trauma
Early viral exanthems (maculopapular rash)	**These conditions cause ecchymoses, petechiae, or purpura, which do not blanch because blood has leaked outside the vessels into surrounding skin and tissue*
Scabies	



Evaluation, cont'd

Description of Secondary Lesions	
Terminology	**Description**
Scaling or hyperkeratosis	Increase in the dead cells on the surface of the skin (stratum corneum)
Desquamation	Skin coming off in scales
Psoriasiform	Large white or silver flakes
Pityriasiform	Branny powdery scale
Lichenoid	Scale tightly adherent to skin surface
Keratotic	Horny scale
Exfoliation	Peeling skin
Maceration	Moist peeling skin
Verrucous	Warty skin
Excoriation	Excavations dug into skin through mechanical means such as rubbing, picking, or scratching
Lichenification	Thickened, rough skin with accentuation of skin markings due to rubbing or scratching
Crusting	Dried exudate of dried serum, blood, and cellular debris over skin lesion
Ulceration	Full thickness loss of epidermis and dermis
Erosion	Superficial denuded lesion due to partial loss of the epidermis
Fissure	Linear split in epidermis, often just extending into dermis
Scar	Flat fibrous tissue after skin damage
Keloid	Hypertrophied fibrous tissue after skin damage

Associated physical examination findings: Supportive attributes found on physical examination may assist in diagnosing dermatoses. Consider including an examination of nails, hair, scalp, eyelids, eyes, nose, mouth, genitals, and joints, as clinically indicated.[1,2]

Clinical Photography: The practice of dermatology relies on visual information. Clinical photography can aid in the diagnosis, monitoring, and documentation of dermatologic conditions, especially when requesting an eConsult and referring for a teledermatology appointment.

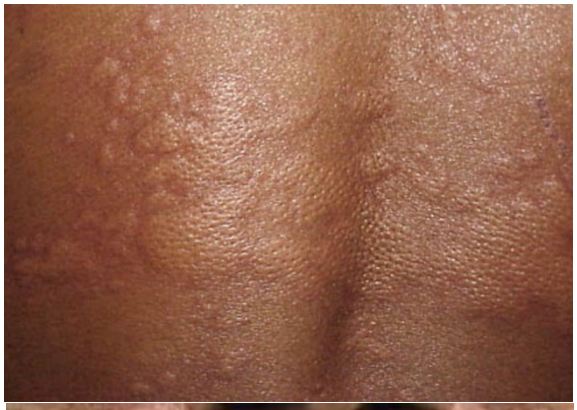
CDCR 7120, Informed Consent for Clinical Photography/Digital Imaging- English
CDCR 7120, Informed Consent for Clinical Photography/Digital Imaging- Spanish

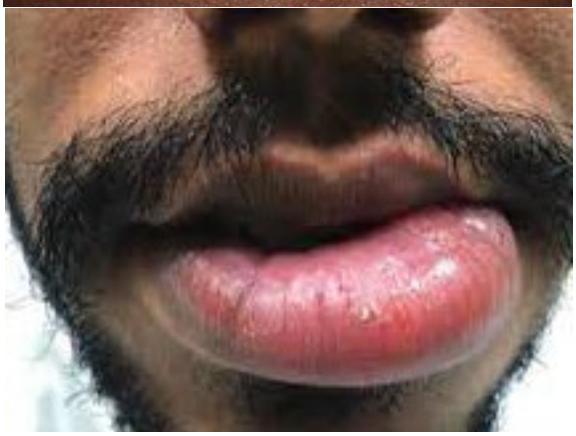
DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

LIFE-THREATENING TYPE I HYPERSENSITIVITY REACTIONS

Type I hypersensitivity reactions, also known as an immediate reaction, involves immunoglobulin E (IgE) mediated release of antibodies against an antigen. This leads to mast cell degranulation and release of histamine and other inflammatory mediators. As a result, there is increased vascular permeability, peripheral vasodilation, and smooth muscle contraction. This section will address severe type I hypersensitivities that are potentially life-threatening.

URTICARIA, ANGIOEDEMA, AND ANAPHYLAXIS





Characteristics

Urticaria, or hives, is characterized by sharpy circumscribed, pruritic wheals and can coexist with angioedema, which is a larger and deeper edematous area involving the dermis and subcutaneous tissue. Acute urticaria due to type I hypersensitivity can also be associated with systemic symptoms, indicating anaphylaxis.

Triggers

- Antibiotics, particularly penicillin, cephalosporins, sulfonamides
- Other medications, such as non-steroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme inhibitors (ACEi), contrast media, immunomodulators, and biologic agents
- Food, such as peanuts, tree nuts, and seafood
- Insect venom, such as ant, venom, bee venom and wasp venom

Evaluation

Anaphylaxis has a rapid onset and is life-threatening. Urticaria and angioedema occur in about 80-90% of cases of anaphylaxis, but it is important to consider that skin and mucosal manifestations are not always present and not directly related to anaphylaxis severity.

Anaphylaxis is highly likely when one of these three criteria are fulfilled:

- Acute onset of an illness within minutes to several hours with involvement of skin, mucosa, or both and at least one of the following
    - Respiratory compromise
    - Reduced blood pressure or associated symptoms of end-organ damage
- Two of more of the following that occur rapidly within minutes to several hours after exposure to a likely allergen
    - Involvement of the skin-mucosal tissue
    - Respiratory compromise
    - Reduced blood pressure or associated symptoms
    - Persistent gastrointestinal symptoms
- Reduced blood pressure after exposure to known allergen within minutes to several hours
    - Systolic blood pressure <90 mmHg
    - Systolic blood pressure drops >30% from patient's baseline

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

HEALTH EQUITY ALERT

A 2021 report looked at health disparities in allergic and immunologic conditions in racial and ethnic underserved populations, and it demonstrated an increasing prevalence of food allergies with the fastest increase in self-reported food allergies among Black patients (2.1% increase per decade vs 1.2% increase among Hispanic patients and 1.0% increase for non-Hispanic White patients). Furthermore, risk factors for fatal drug anaphylaxis included Black race and age 80 yo or older. Accurate documentation of food and drug allergies leads to more timely diagnosis and treatment of these life-threatening type I hypersensitivity reactions. Additionally, all patients should be educated and equipped to recognize and manage their food allergies and drug allergies, which impacts disease morbidity and mortality.

Acute urticaria typically lasts less than 6 weeks. Wheals in a fixed location for more than 24 hours suggest the possibility of urticarial vasculitis and warrant a skin biopsy. Chronic idiopathic urticaria for which no trigger can be identified often requires further testing such as skin prick testing (SPT), serum radioallergosorbent testing (RAST), or skin patch testing. After an acute event when the patient is stabilized, consider referring patients for allergy testing.

SPTs, which are also known as scratch tests, are the cheapest and most effective method to diagnose IgE-mediated type 1 hypersensitivity reactions. However, skin testing should **not** routinely be performed in patients who are at high risk for an anaphylactic reaction to testing, patients who have experienced a recent anaphylactic event, patients taking medications that may interfere with the treatment of anaphylaxis, or patients with atopic dermatitis. In vitro allergy testing to measure specific serum IgE antibodies should be the initial diagnostic modality in such patients. Consider comanagement with allergy and immunology specialists regarding the most appropriate allergy testing.

Treatment

Triage suspected anaphylactic reactions with urgency since patients are at risk for rapid deterioration with the development of angioedema or anaphylaxis.

- Assess the patient's vital signs and level of consciousness. If the patient is clinically unstable or has altered level of consciousness, initiate EMS transfer to higher-level of care (HLOC).
- Maintain a patent airway. Perioral edema, stridor, and angioedema are very high risk, and obtaining a definitive airway is imperative. Delay may reduce the chances of successful intubation as continued swelling occurs, increasing the risk for a surgical airway.
- Monitor cardiac rate, cardiac rhythm, and oxygen saturation.
- Establish IV access with large bore needle and infuse 0.9% sodium chloride intravenous solution at 30 cc/hour. Consider 1-2 L IV bolus if patient is hypotensive for fluid resuscitation.
- Administer epinephrine injection 0.3-0.5 mg IM, which may be repeated every 5 minutes x 2 as needed for no clinical improvement in symptoms or inadequate response.
- Decontaminate the offending agent, if possible, to prevent continued exposure and clinical worsening. Remove any stingers, if present.

No non-life-threatening urticaria, treatment includes elimination of known causes, antihistamines (H_1 and H_2 blockers), oral corticosteroids for acute flares, and, in refractory cases, immunosuppressants, such as sulfasalazine and cyclosporine. Consider comanagement with allergy and immunology specialists regarding the most appropriate therapies for the patient. Lastly, consider developing an Allergy and Anaphylaxis Emergency Plan with the patient. All food and drug allergies should be documented and updated in EHRS.[3,4]

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

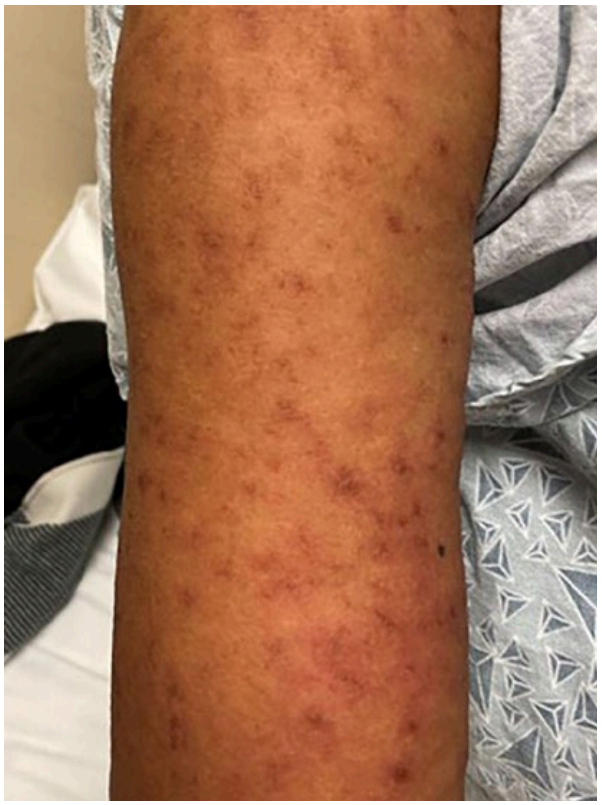
LIFE-THREATENING TYPE IV HYPERSENSITIVITY REACTIONS

Cutaneous adverse drug reactions (CADR) can be caused by type I hypersensitivity reactions, type IV hypersensitivity reactions, or nonimmune-related drug reactions. These reactions can range from mild to life-threatening. Type IV hypersensitivity reactions include exanthematous drug eruptions (EDE), erythema multiforme (EM), Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN), and drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome, which will be addressed in this section.

HEALTH EQUITY ALERT

Genetic predispositions for life-threatening type IV hypersensitivity reactions can be determined by personal and family history. For example, the HLA-B*5801 allele is associated with allopurinol hypersensitivity reactions. Consider testing before starting certain medications for select high-risk populations.

EXANTHEMATOUS DRUG ERUPTIONS



Characteristics

Exanthematous drug eruptions (EDE) are predominantly caused by type IV hypersensitivity reactions, and the most common type of EDE is maculopapular rash (MPR), which is characterized by erythematous macules and papules that may coalesce to form plaques. This MPR typically starts on the face, neck, and upper trunk, then extends symmetrically toward the lumps.

Triggers

- Acetaminophen
- Allopurinol
- Antibiotics, like penicillin, cephalosporins, fluoroquinolones, sulfonamides, macrolides, minocycline
- Antiseizure medications (ASM), like carbamazepine, phenytoin, phenobarbital, lamotrigine, valproate
- Biologics
- NSAIDs

Evaluation

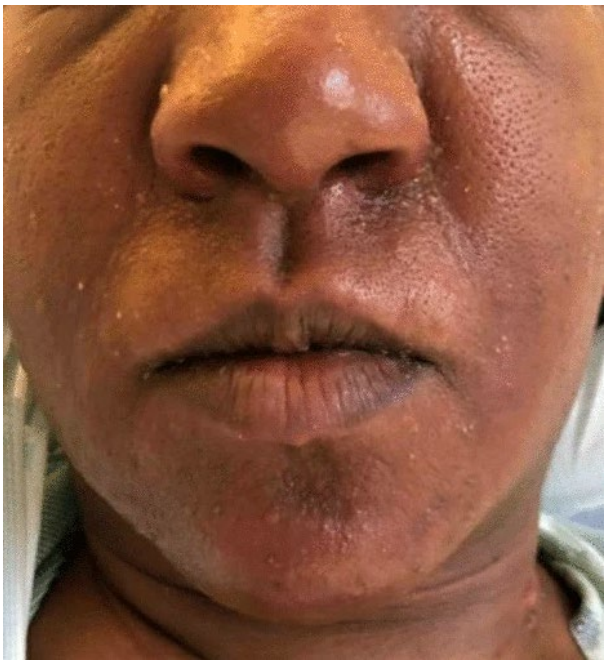
The diagnosis of EDE is primarily clinical. The rash develops hours to two weeks after the offending drug is first given, and the timing can differ if previously sensitized. MPR has no mucosal involvement, no blistering, fever, or lymphadenopathy. A severe CADR, such as DRESS, SJS, and TEN, may start as MPR, but patients may also have mucosal involvement, blistering, and/or systemic symptoms.

Treatment

The first approach to any CADR is prompt withdrawal and future avoidance of the offending medication(s). Supportive treatment with a topical medium- to high-potency corticosteroid and an oral second-generation antihistamine, such as cetirizine or loratadine, can provide symptomatic relief of skin lesion and pruritis.[4]

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

DRUG REACTION WITH EOSINOPHILIA AND SYSTEMIC SYMPTOMS



Characteristics

DRESS syndrome, another type IV hypersensitivity reaction, is a severe, widespread CADR with systemic involvement associated with visceral organ dysfunction, lymphadenopathy, eosinophilia, and atypical lymphocytosis.

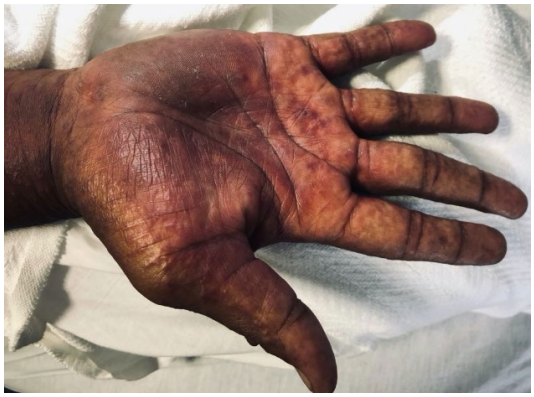
Triggers

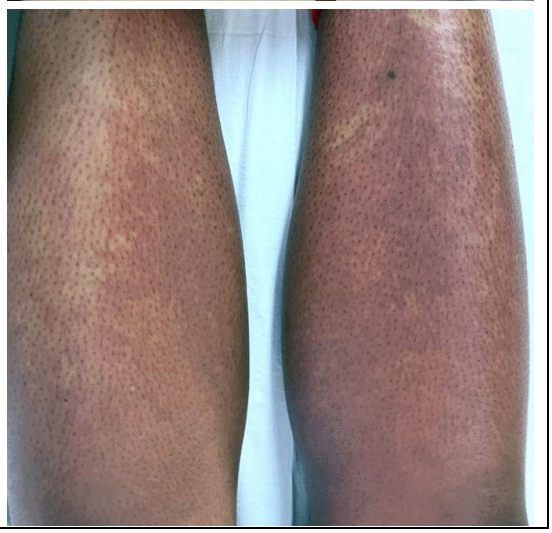
The risk of developing DRESS syndrome varies from drug to drug. For high-risk ASMs, the incidence of DRESS is estimated to be 1 in 1000 to 1 in 10,000 exposures.

- Allopurinol
- Antibiotics, like penicillin, cephalosporins, dapsone, fluoroquinolones, sulfonamides, macrolides, minocycline, trimethoprim-sulfamethoxazole (TMP-SMX), vancomycin
- Antituberculosis agents, like rifampicin, ethambutol, isoniazid, pyrazinamide
- Aromatic ASMs, like carbamazepine, phenytoin, phenobarbital, lamotrigine, oxcarbazepine

Evaluation

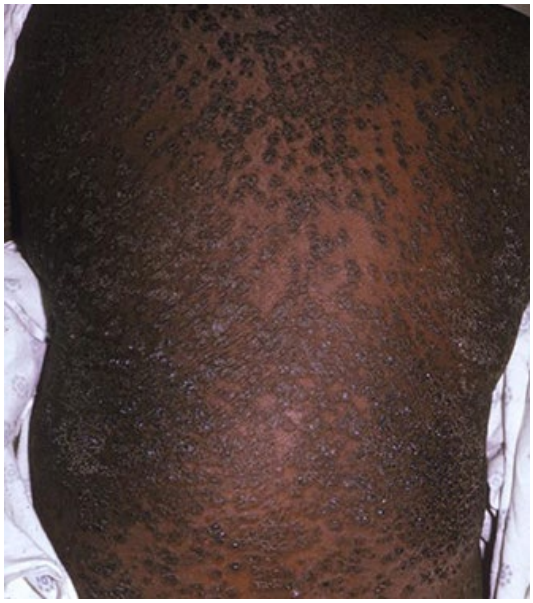
The clinical presentation of DRESS syndrome is heterogeneous, and the disease course is typically prolonged. Despite the cessation of the offending drug, flares of disease may continue to occur. The latency between drug initiation and onset of disease is prolonged, typically between 2-8 weeks.





- Cutaneous lesions are polymorphic and evolving. DRESS starts as MPR that may be diffusely pruritic. Coalescing erythema (erythroderma) may be difficult to assess on skin of color. Patient may also present with facial edema, purpura, infiltrated plaques, pustules, exfoliative dermatitis, and target-like lesions
- Mucosal involvement is typically mild
- Systemic symptoms and laboratory abnormalities include
    - Fever (75-90%)
    - Lymphadenopathy (54-65%)
    - Eosinophilia > 700/microL (82-95%)
    - Leukocytosis (95%)
    - Neutrophilia (78%
    - Lymphocytosis (25-52%)
    - Monocytosis 69%)
    - Atypical lymphocytes (35-67%)
    - Lymphopenia (45%)
    - Thrombocytopenia (25%)
    - Liver involvement: cholestatic (37%), hepatocellular (19%), or mixed (27%) with liver enzymes more than 10 times the upper limit of normal in up to half of DRESS syndrome cases

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY



    - Kidney involvement: range from proteinuria to kidney failure, acute interstitial nephritis (AIN) (10-30%)
    - Pulmonary involvement with shortness of breath and dry cough, which may be due to acute interstitial pneumonitis, lymphocytic interstitial pneumonia, pleuritis, and acute respiratory distress syndrome
- Notably, DRESS syndrome can be confused with EDEs, which can have mild systemic symptoms, like a low-grade fever, pruritic, and mild eosinophilia. However, EDEs do not have visceral involvement.

Treatment

The first approach to any CADR is prompt withdrawal and future avoidance of the offending medication(s). Because of the heterogeneity of clinical manifestations, management is based on the severity of DRESS syndrome and organ involvement.

Mild DRESS Syndrome: Defined as DRESS with or without modest liver involvement (elevation of liver transaminases < 4 times the upper limit of normal, **in the absence of** clinical, laboratory, or imaging evidence of renal, pulmonary, or cardiac involvement

- Can be managed in the outpatient setting with symptomatic treatment and close clinical and laboratory monitoring for possible organ involvement.
- **Identify and discontinue causative medication.**
- Supportive treatment with a topical high- to super high-potency corticosteroid provides symptomatic relief of skin lesion and pruritus.
- Gentle skin care with emollients and warm baths with wet dressing.

Moderate to Severe DRESS Syndrome:

- Widespread rash and severe systemic symptoms require transfer to HLOC for evaluation and treatment.
- **Identify and discontinue causative medication.**
- Upon patient return from HLOC when clinically stable, continue management per recommendations by dermatologist and other specialists (e.g. hepatologist, nephrologist, pulmonologist).

Prevention of Relapses:

- Avoid offending medication and cross-reacting medications. For example, patients previously treated with aromatic ASMs should be treated with nonaromatic agents instead.
- Avoid routine and unnecessary exposure to antibiotics and other medication during the acute phase.
- Undergo long-term monitoring for the development of autoimmune sequelae, such as autoimmune thyroiditis, vitiligo, alopecia areata or alopecia universalis, autoimmune hemolytic anemia, lupus, or type 1 diabetes.[4]

ERYTHEMA MULTIFORME

Characteristics

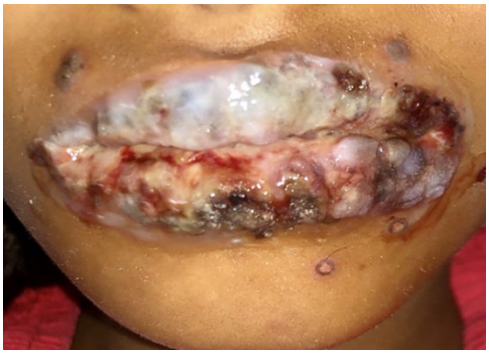
Erythema multiforme is a cutaneous and mucosal type 4 hypersensitivity reaction that presents with papular, bullous, and necrotic lesions. Typical lesions are symmetrical, dusky, raised target lesions.

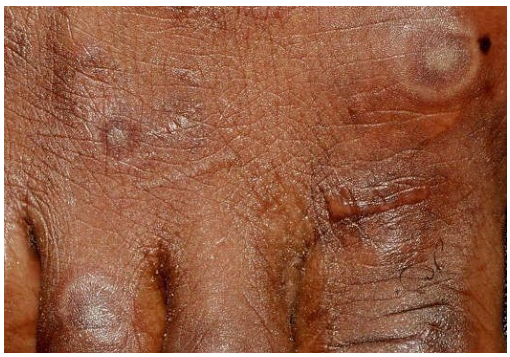
Most cases of erythema multiforme are due to infection. However, erythema multiforme can also be caused by medications in fewer than 10% of cases. Patients who are immunosuppressed, such as patients living with HIV and patients prescribed corticosteroids, are predisposed to developing erythema multiforme.

Most cases of erythema multiforme are mild with minimal to no mucosal involvement and are designated erythema multiforme minor (EMm). Rarely, erythema multiforme can be life-threatening and is called

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

erythema multiforme major (EMM). EMM presents with typical target lesions as well as involvement of multiple mucosal and ophthalmic sites causing bullae and erosions. Drug-related EMM have widespread and severe involvement of the oral mucosa and lips. Bullae rupture causing hemorrhagic pseudomembrane of the lips leading to bloody encrustation and ulceration.



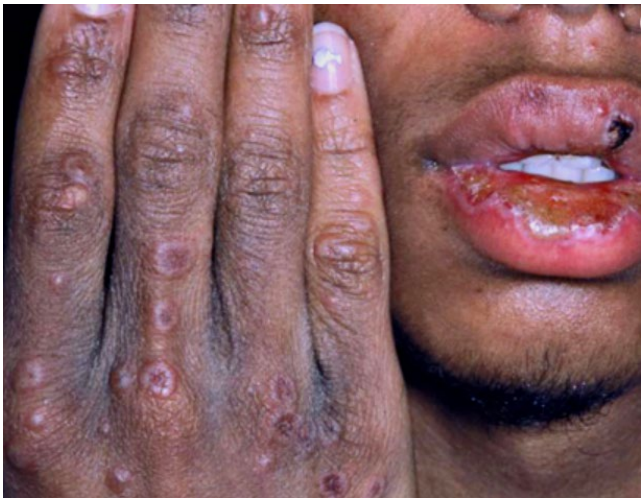


Triggers

- Penicillin
- Cephalosporins
- Macrolides
- Sulfonamides
- Anti-tuberculosis agents
- Chemotherapeutic agents
- Poison ivy
- Herpes simplex virus
- Ebstein Barr virus
- *Mycoplasma pneumoniae*
- Histoplasmosis

Evaluation

Erythema multiforme is clinically diagnosed with cutaneous lesions that usually appear over the course of three to five days after exposure or trigger. Although the skin lesions do not scar, post inflammatory hyperpigmentation may remain for months after resolution, particularly in patients with dark skin. Typical target lesions are defined as individual lesions less than 3 cm in diameter with a regular round shape, well-defined border, and at least three different zones. Raised atypical targets are round, edematous, palpable lesions, with only two zones and/or a poorly defined border. Skin lesions are usually asymptomatic, but patients may endorse itching or burning.



EMM is distinguished from EMm with EMM presenting with extensive mucosal involvement of multiple sites of the body as well as the presence of systemic symptoms such as fever and arthralgia. Laboratory tests such as CBC, CMP, and CRP are not specific.

Given the clinical and histopathologic similarities, EMM and Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis (SJS/TEN) should be distinguished as different and distinct conditions, usually with an oral biopsy, if recommended by a specialist. Diagnosis of EM Major would be appropriate in a patient with the following clinical features:

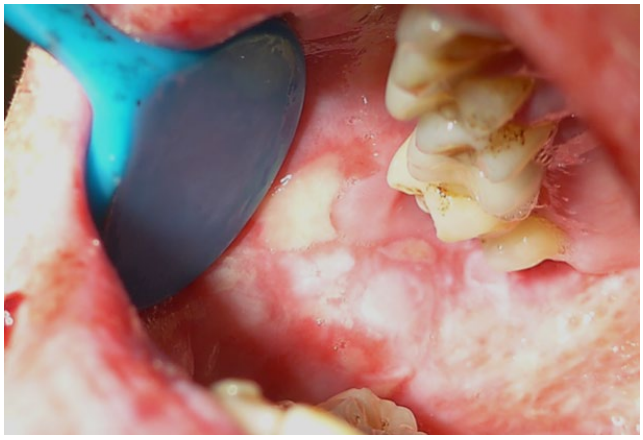
- A history of recent infection strongly suggests EMM. Although medications are associated with 10% of EMM, medications are the leading cause of SJS/TEN.
- A prodrome of febrile illness and malaise can be seen in EMM but is not specific and can be seen more often in SJS/TEN.
- EMM mostly does not have skin detachment and, if present, is less than 10% of the total body surface area. Conversely, SJS/TEN is commonly associated with skin detachment.
- EMM is characterized by typical target-like or raised atypical targets lesions.
- EMM skin lesions are symmetrical and localized, which start from the extremities and could move centripetally.
- Comparing to SJS/TEN, recurrences are more common with EMM.

Consider referral to dental to comanage patients.

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

Treatment

Although EMm is self-limited, EMM or recurrent cases of erythema multiforme require consultation with dermatology and systemic therapy, usually in at HLOC. Symptomatic treatment for EMm is the mainstay of therapy with topical steroids and antihistamines.



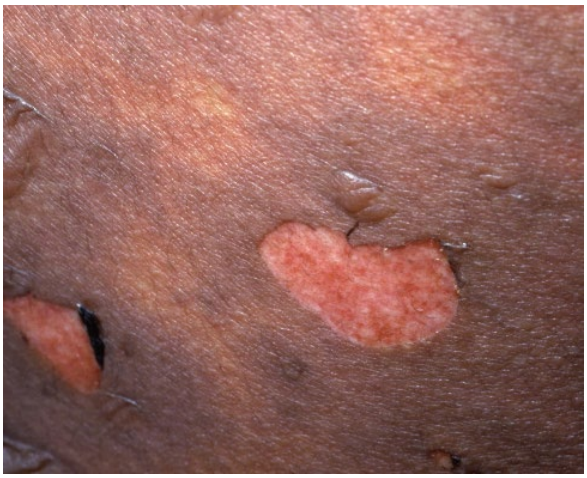
Consider referral to dental to comanage patients. For limited oral mucosal involvement, a mouthwash containing equal parts of viscous lidocaine 2% with aluminum hydroxide and magnesium hydroxide mixture (e.g., Maalox®) as a swish-and-spit, can be used as needed, up to four times per day to alleviate pain. Dexamethasone (0.5 mg/5 mL) oral elixir 4 times/day swish-and-spit can be used for diffused ulcers. If the ulcers are limited to a small area, fluocinonide 0.05% gel is applied two to three times per day.

For extensive or severe oral mucosal involvement that prevents sufficient oral intake, send patient to HLOC. Consider antiviral suppression therapy, such as acyclovir 400mg twice a day, for recurrent EM associated with HSV.[4]

STEVENS-JOHNSON SYNDROME AND TOXIC EPIDERMAL NECROLYSIS

Characteristics

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are considered a disease continuum, distinct from erythema multiforme. SJS/TEN are severe cutaneous adverse reactions, usually to medications, that cause extensive detachment of the epidermis and necrosis. Mucous involvement occurs in more than 90% of cases of SJS/TEN. Overall mortality rate of SJS/TEN during an acute episode is 23% and 34% at one year.



Patient at increased risk of SJS/TEN are patients living with HIV (PLWH), patients with connective tissue disease, patients with cancer, older patients (age ≥65 years old), and patients with impaired renal function. Symptoms of SJS/TEN can start 1 week to 4 weeks after starting a causative medication, which are listed below. Less common triggers are infection, usually when drug causality is unclear.

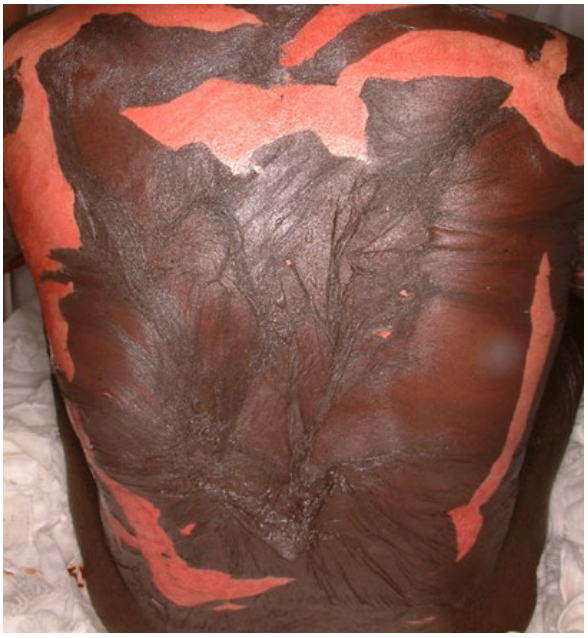
Triggers

- Acetaminophen
- Allopurinol
- Antibiotics, like penicillin, cephalosporins, fluoroquinolones, sulfonamides, macrolides, minocycline
- Antiseizure medications (ASM), like carbamazepine, oxcarbazepine, phenytoin, phenobarbital, lamotrigine, valproate
- Biologics
- Cyclooxygenase-2 (COX-2) NSAIDs
- Immune checkpoint inhibitors (ICI), like ipilimumab, pembrolizumab, nivolumab, atezolizumab
- *Mycoplasma*

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

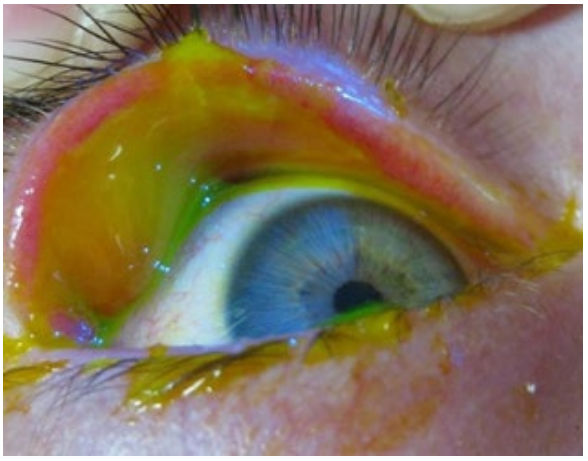
Evaluation

SJS/TEN is clinically diagnosed, and symptoms include malaise, fever, myalgia, sore throat, and conjunctivitis, which may precede or occur concurrently with mucocutaneous presentation.



The initial cutaneous presentation of SJS/TEN may mimic an exanthematous rash. Cutaneous lesions start on the face and thorax before spreading to other areas and are symmetrically distributed. SJS/TEN skin lesions are widespread, starting from the neck, face, and upper torso that extend centrifugally. Early lesions typically begin with ill-defined, coalescing macules, which evolve over the course of one to two days to develop dusky purpuric spots, atypical targets (atypical targets have two rings instead of the three typically seen in target lesions of erythema multiforme), and flaccid bullae.

Nikolsky's sign is almost always found in SJS/TEN, as well as pemphigus and staphylococcal scalded skin syndrome (SSSS). This sign differentiates intraepidermal blisters from subepidermal blisters. Nikolsky's sign is done by applying lateral pressure by a thumb or a finger in the perilesional skin, affected skin, or normal skin, which results in a force that dislodges upper layers of epidermis from lower layers.



All mucosal surfaces can be involved during the acute phase of the disease and often include the oro/nasopharyngeal, buccal, and anogenital mucosa. Ocular involvement is common and may present at conjunctival hyperemia, pseudomembrane formation, or complete corneal epithelial defect.

Multiorgan dysfunction can occur. Acute kidney injury, usually with proteinuria, is common and often requires renal replacement therapy. During the acute phase of SJS/TEN, respiratory complications, such as sloughing of the bronchial epithelium, pneumonia, pulmonary edema, and atelectasis, are frequent. Other abnormalities can include liver injury, gastrointestinal involvement, and hematologic abnormalities.

SJS/TEN is commonly associated with skin detachment. SJS is the less severe condition comparing to TEN, in which skin detachment is <10 % of the total body surface area. TEN involves detachment of >30% of the TBSA. When skin detachment is between 10-30% of TBSA, this is diagnosed as overlap SJS/TEN.

Unlike the raised lesions seen in erythema multiforme, SJS/TEN lesions are commonly flat atypical targets and macules defined as round lesions with only two zones and/or a poorly defined border. Also, SJS/TEN lesions might present as purpuric macules with or without blisters. In general, blisters often occur on all or part of the SJS/TEN lesions.

Treatment

During the acute phase, SJS/TEN worsens progressively over five to seven days with extensive cutaneous detachment and mucositis, so HLOC is required. Due to extensive skin detachment during the acute phase, patients are prone to fluid and electrolyte imbalances, sepsis, organ decompensation, and death. There is no established pharmacologic treatment for SJS/TEN, so supportive care is the cornerstone of management, which includes wound care, fluid and electrolyte management, nutritional support, pain control, infection prevention, ocular care, and organ support.

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

The chronic phase occurs during the convalescent and recovery stage of SJS/TEN. Management principles include the screening and treatment of complications to maintain life quality. Long-term sequelae include cutaneous, mucosal, ocular, visceral, and psychologic complications, which are increasingly reported as survival improves. Cutaneous sequelae include post inflammatory hypo- or hyperpigmentation, scarring, eruptive nevi, telogen effluvium, and chronic pruritus. Ocular sequelae include dry eye, photophobia, trichiasis, neovascularization of the cornea, keratitis, symblepharon, and corneal scarring leading to visual impairment, so referral to specialists may be needed. Oral discomfort, gingival inflammation, caries, and periodontal disease should be followed by dentistry. Long-term urogenital and pulmonary complications should be closely followed and managed, especially since a third of patients who survive SJS/TEN report chronic pain at mucocutaneous sites. Consider referral to dental to comanage patients.

Patients who survive SJS/TEN attributed to a medication must be educated about future avoidance and understand that re-exposure to the culprit drug and chemically-related agents may be fatal. Furthermore, because human leukocyte antigen (HLA) groups are associated with carbamazepine-, phenytoin-, phenobarbital-, and allopurinol-induced SJS/TEN, patients and family members of patients should be alerted against the use of the same medication classes.

Unfortunately, survivors of SJS/TEN often exhibit avoidance behaviors due to the significant psychological impact of the condition. One study demonstrated that >60% of patients were fearful of taking newly prescribed medications, and 30% of patients avoided taking the prescribed medications for their diagnosed medical conditions. Post-traumatic stress disorder (PTSD) affects 20% of patients who survived SJS/TEN 6 months after the initial episode. Depression and anxiety may affect up to 30-50% of patients who survived SJS/TEN. Therefore, consider a referral to mental health for these patients.[4]

DERMATOLOGIC MANIFESTATIONS OF INFECTION

Most infections with dermatological manifestations are self-limited, but the following clinical scenarios are associated with life-threatening systemic symptoms. The majority of infectious emergencies in dermatology are due to a bacterial pathogen. However, select viruses, fungi, and parasites can also cause severe disease. Therefore, prompt recognition and transfer to higher level of care for treatment are paramount.

This section highlights the main cutaneous patterns, individuals at risk, diagnostic modalities applicable at institutions, and treatment options for each infectious emergency.

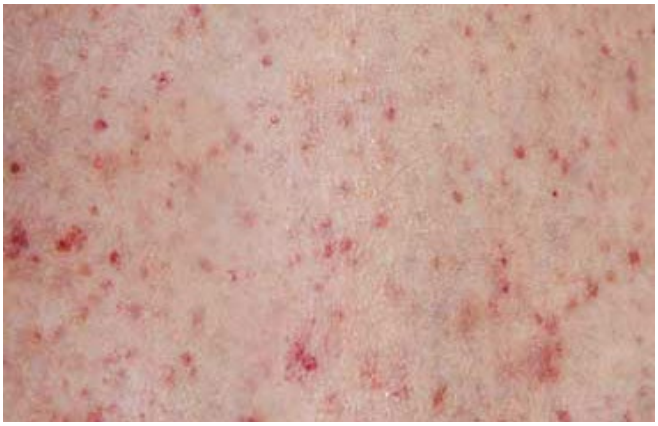
Although mentioned in this care guide, the evaluation and treatment of skin and soft tissue infections (SSTI) as well as chronic wounds are not detailed. Please refer to the Skin and Soft Tissue Infection Care Guide and the Chronic Wound Management Care Guide for more information.

Also refer to the Sexually Transmitted Infection Care Guide for further guidance on assessment and management.

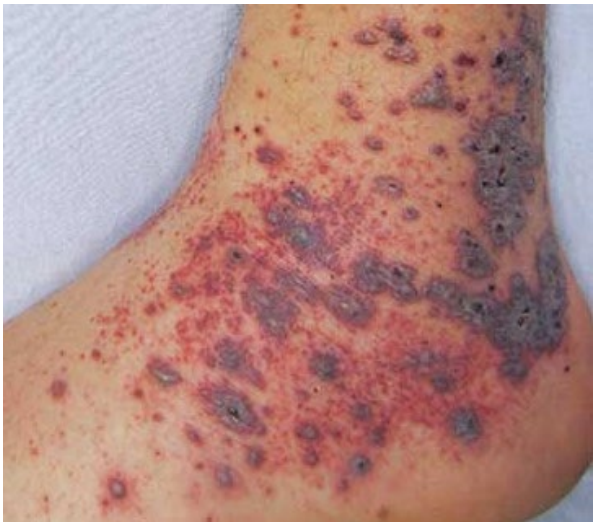
ACUTE MENINGOCOCCEMIA

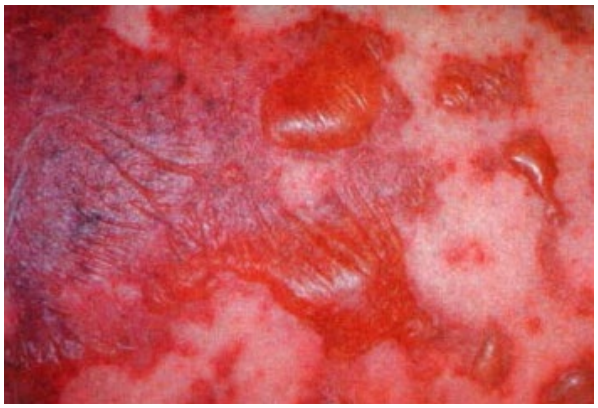
Characteristics

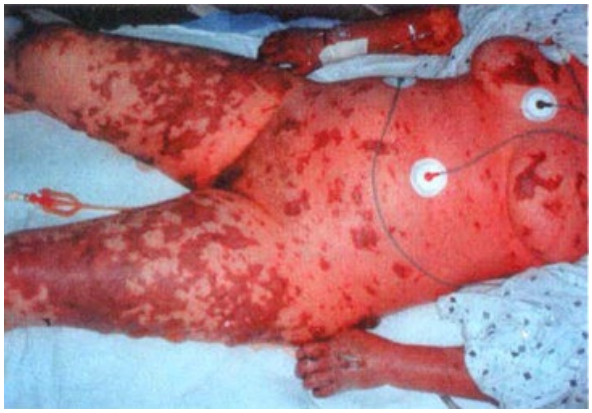
Neisseria meningitidis (*Meningococcus*) is a life-threatening bacterial infection that can manifest as meningitis or septicemia, or more often a combination of both. Meningococcal disease is a serious public health threat given the seriousness of the illness, its disabling sequelae, and its potential for epidemic spread. The disease is a concern during mass gatherings, which provide conditions that facilitate transmission of *N. meningitidis*. Patients most likely to be carriers are adolescents and young adults, not due to their age itself but rather, due to social behaviors that increase the risk of transmission such as living in dormitories or enclosed spaces, smoking, and frequent intimate oral activities. In fact, it's estimated that up to 23% of 19-year-olds are carrying the bacteria at any given time.



DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY







Evaluation

The characteristic rash of acute meningococcemia presents as petechiae and purpura in approximately 50-75% of cases. Cutaneous lesions are tender, well-defined, indurated, retiform purpura, which can progress to vesicles and bullae with the formation of necrosis and eschar.

Fulminant meningococcemia presents with purpura fulminans, widespread ecchymoses, and hemorrhagic bullae. Purpura fulminans is a severe complication of acute meningococcemia and is characterized by acute cutaneous hemorrhage with necrosis and bullae formation due to vascular thrombosis.

Other symptoms and signs include:

- Fever
- Severe headache
- Stiff neck
- Photophobia
- Loss of appetite, nausea, and vomiting
- Arthralgias and myalgias
- Altered mental status
- Hemodynamic compromise due to sepsis

Management

Prompt diagnosis and treatment is critical, so transfer patients with suspected acute meningococcemia to HLOC. Effective antibiotics should be administered promptly due to risk of severe morbidity and death. Empirical therapy for suspected meningococcal disease should include an extended-spectrum cephalosporin, such as cefotaxime or ceftriaxone. Once the microbiologic diagnosis is established, definitive treatment can be continued with an extended-spectrum cephalosporin (cefotaxime or ceftriaxone). Alternatively, if susceptibility of the meningococcal isolate to penicillin is confirmed, treatment can be switched to penicillin G or ampicillin.

Prevention

CDC recommends meningococcal vaccination for patients at increased risk for meningococcal disease.

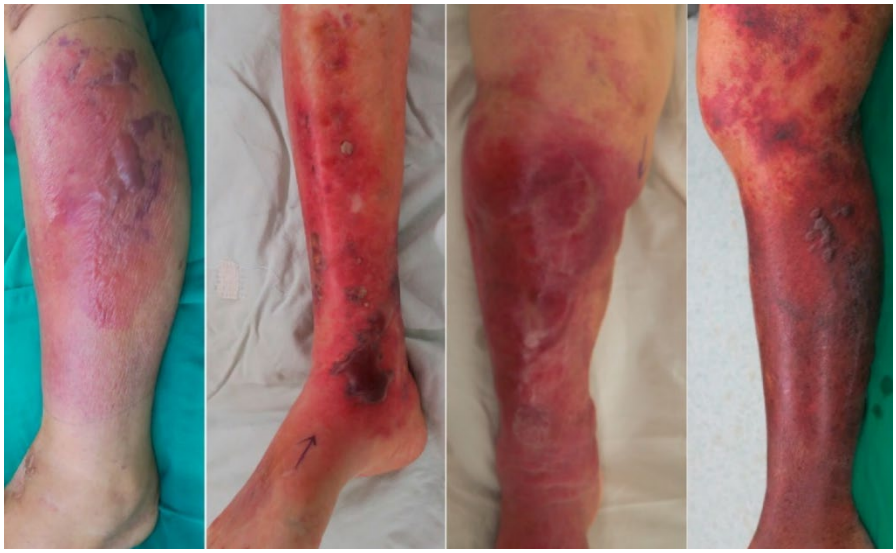
- People living together in close quarters, such as dormitories
- People who are immunocompromised, like HIV or persistent complement component deficiencies
- People >85 years old
- People with anatomic or functional asplenia
- People who receive complement inhibitors, like eculizumab (Soliris®), ravulizumab (Ultomiris™)

Additionally, CDC recommends prophylaxis for close contacts of patients with meningococcal disease, regardless of vaccination status. Rifampin (4 oral doses in 48 hours), ciprofloxacin (single oral dose), or ceftriaxone (single injection) are first-line antibiotics for meningococcal prophylaxis. A single oral dose of azithromycin has also been used in areas with ciprofloxacin-resistant strains.[5]

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

NECROTIZING FASCIITIS

Please refer to the Skin and Soft Tissue Infection Care Guide for more detailed information on necrotizing fasciitis, which is an aggressive skin and soft tissue infections (SSTI) that cause necrosis of the muscle fascia and subcutaneous tissues. A thorough examination of the skin will often detect subtle evidence of a soft tissue infection such as localized swelling, tenderness, or the more distinctive violaceous bullae that may be seen in necrotizing fasciitis.



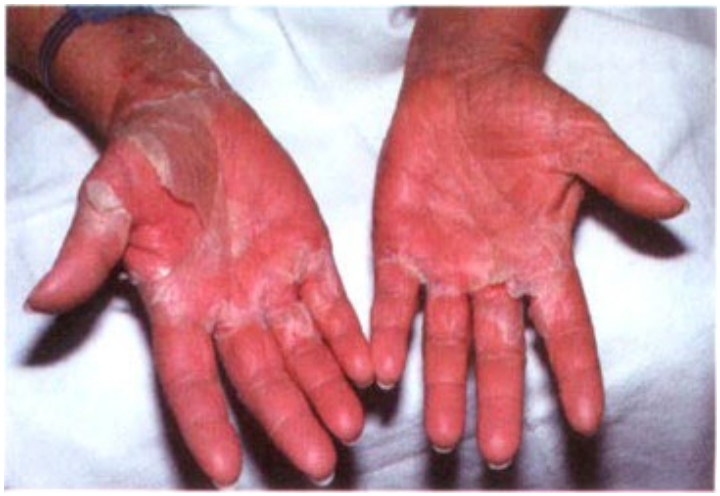
Necrotizing fasciitis can be cause by group A streptococcus, *Vibrio vulnificus*, *Clostridium perfringens*, *Escherichia coli*, and other bacteria. Group A streptococcus is the most common bacteria to cause necrotizing fasciitis.

Necrotizing infections more commonly present with excruciating pain out of proportion to presenting symptoms and systemic septic signs.

TOXIC SHOCK SYNDROME

Characteristics

Toxic shock syndrome (TSS) is a clinical illness characterized by rapid onset of fever, rash, hypotension, and multiorgan system involvement, which can be caused by *Staphylococcus aures*, group A streptococci (*Streptococcus pyogenes*), or Clostridium sordellii. Women and patients with a uterus who develop staphylococcus TSS are more likely to have used tampons with high absorbency, used tampons continuously for more days of their cycle, and kept a single tampon in place for a longer time. Nonmenstrual staphylococcus TSS can occur in a variety of clinical circumstances, including surgical and postpartum wound infections, mastitis, septorhinoplasty, sinusitis, osteomyelitis, arthritis, burns, cutaneous and subcutaneous lesions (especially of the extremities, perianal area, and axillae), respiratory infections following influenza, and enterocolitis.

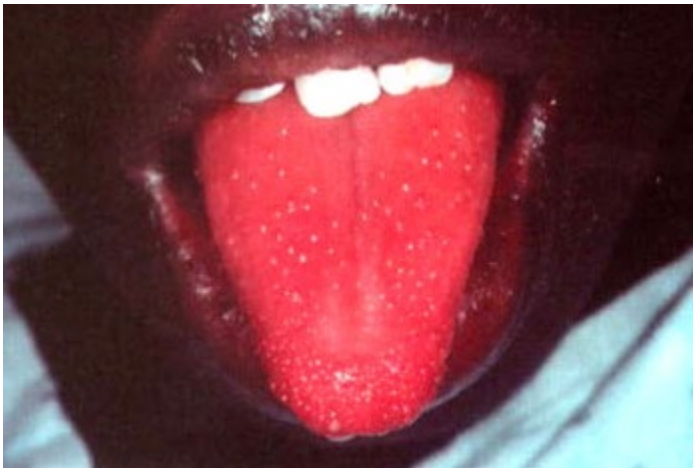


Most reported cases of staphylococcus TSS have been due to methicillin-susceptible *S. aureus* (MSSA). However, as rates of infection due to methicillin-resistant *S. aureus* (MRSA) have increased, cases of staphylococcus TSS due to MRSA have also emerged. *S. aureus* exotoxins are superantigens that cause massive cytokine production.

Streptococcal TSS is caused by infection with group A streptococcus (*Streptococcus pyogenes*) and the production of streptococcal pyrogenic exotoxins A, *B*, and C. This syndrome occurs most commonly in the setting of invasive soft tissue infections, such as necrotizing fasciitis, myonecrosis, and cellulitis. The portal of entry for the bacteria can occurs through a burn site, laceration, surgical incision, pressure ulcer, childbirth trauma, or varicella lesion.

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

Evaluation



Prodromal features of patients with staphylococcal toxic shock syndrome include fever, malaise, myalgia, nausea, vomiting, diarrhea, and prominent confusion.

Cutaneous signs of streptococcal toxic shock syndrome may be subtle or absent. The cutaneous signs include a sunburn like diffuse macular erythroderma followed by desquamation, especially of the hands and feet, within 5 to 14 days. Desquamating erythroderma may be present but is less common than in staphylococcal toxic shock syndrome. Conjunctival injection, mucosal hyperemia (oral and genital), and a strawberry tongue. are appreciable and are important diagnostic signs in deeply pigmented patients in whom the erythroderma may be subtle or overlooked. Less frequent manifestations are edema of the hands and feet, petechiae, and delayed loss of nails and hair.

Multiorgan involvement may include gastrointestinal, hepatic, musculoskeletal, renal, cardiopulmonary, central nervous system, and metabolic findings.

Streptococcal toxic shock syndrome also presents with fever, hypotension, cutaneous findings, and severe local pain. Both dramatic and rapid progression of local and systemic findings are hallmarks of streptococcal toxic shock syndrome. The pain is typically localized to an extremity and is often disproportionate to the findings on examination.

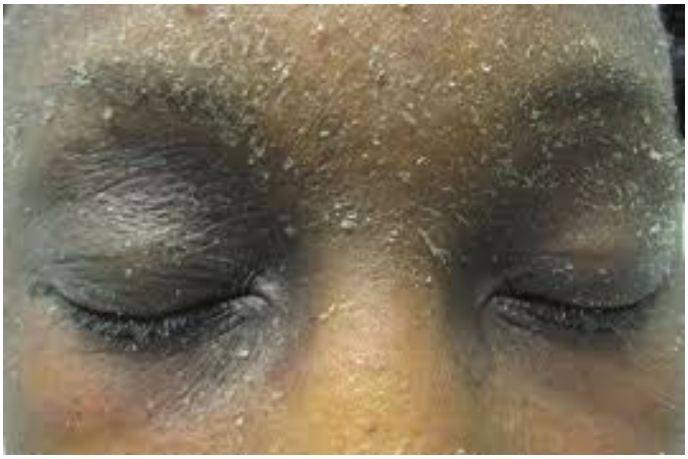
Treatment

Prompt diagnosis and treatment is critical, so transfer patients with suspected TSS to HLOC. Treatment involves identification and removal of the source of *S. aureus* (surgical debridement or drainage or removal of nasal packing or tampon) or *Streptococcus* (surgical debridement or drainage), initiation of effective antibiotic therapy, and appropriate supportive care.

Tampons and barrier contraceptives should be avoided in women and patients with a uterus in whom seroconversion does not occur after an acute illness.[6]

STAPHYLOCOCCAL SCALDED SKIN SYNDROME

Characteristics



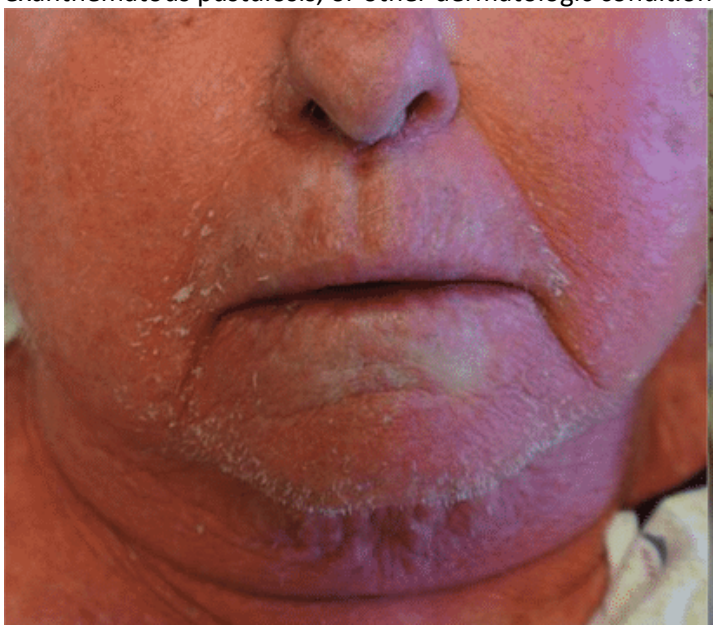
Staphylococcal scalded skin syndrome (SSSS) is a systemic cutaneous infection caused by toxin-producing staphylococcal species, which presents as extensive blistering and desquamation. SSSS usually affects children because adults have neutralizing antibodies and mature kidney function, which allow for efficient clearance of exotoxins from the bloodstream. However, consider SSSS in adults with immunosuppression, including people living with HIV, patients with advanced malignancies, or those with severe kidney disease.

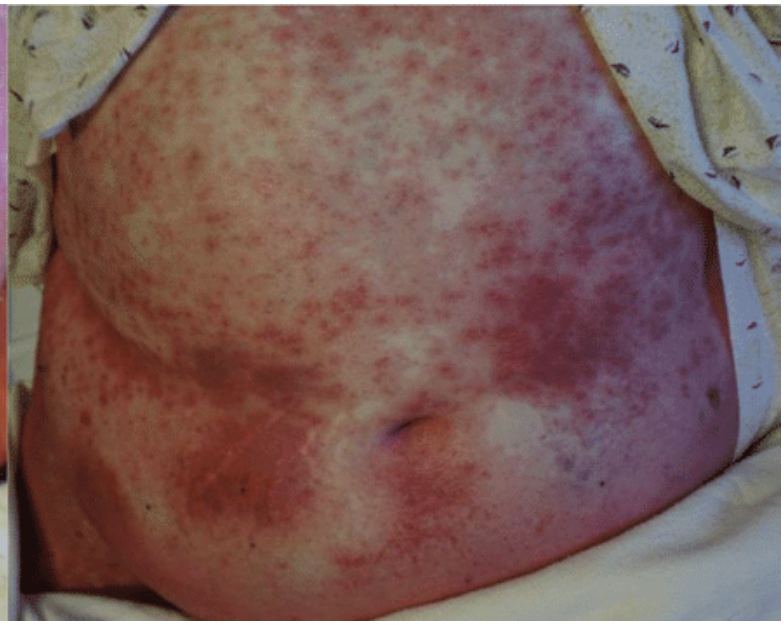
Evaluation

SSSS initially presents with irritability, fever, and malaise, followed by the rapid development of a tender, erythematous desquamative rash on the face and flexures, such as the groin, axillae, and neck, within 24 to 48 hours. Subsequently, large, fragile, and potentially purulent blisters form bullae, exhibiting a positive Nikolsky sign. Progressive desquamation and healing without scarring occur over the next 2 weeks.

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

The evaluation of SSSS is clinical and should be distinguished from other causes of blistering and desquamation, such as Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis, acute meningococcemia, toxic shock syndrome, acute generalized exanthematous pustulosis, or other dermatologic conditions.





Management

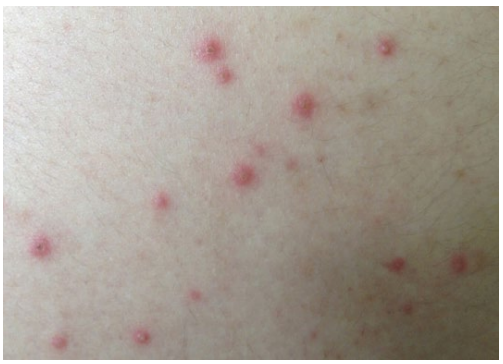
Prompt diagnosis and treatment is critical, so transfer patients with suspected SSSS to HLOC. Treatment involves identification and removal of the source of staphylococcus, initiation of effective antibiotic therapy, and appropriate supportive care.

Additional antibiotics with *Pseudomonas* coverage should be initiated if concern for a secondary bacterial skin infection arises. Acetaminophen and opioids may be needed for pain control, but NSAIDs, such as ibuprofen, should be avoided due to the risk of kidney impairment. Please refer to the Skin and Soft Tissue Infection Care Guide for more information.[7]

DISSEMINATED HERPES SIMPLEX

Characteristics

Primary herpes simplex virus (HSV) infection may rarely lead to widespread vesicular eruptions in the immunocompetent host or visceral disease (e.g., fulminant hepatitis, encephalitis, myocarditis) with or without mucocutaneous lesions. This can occur with either HSV-1 or HSV-2. Typical oral and/or genital lesions occur in only 30 percent of patients, although sometimes scattered skin lesions can be identified as widespread eruption of vesicles, pustules, and erosions.



Patients at risk for disseminated HSV are patients taking steroids, transplant recipients, patients with burn-related injuries, persons living with HIV, those with cancer or myelodysplastic syndromes, and pregnant people. In addition, babies born to people with genital HSV-1 infection may be at risk of neonatal HSV infection, particularly if genital HSV-1 is acquired during pregnancy.

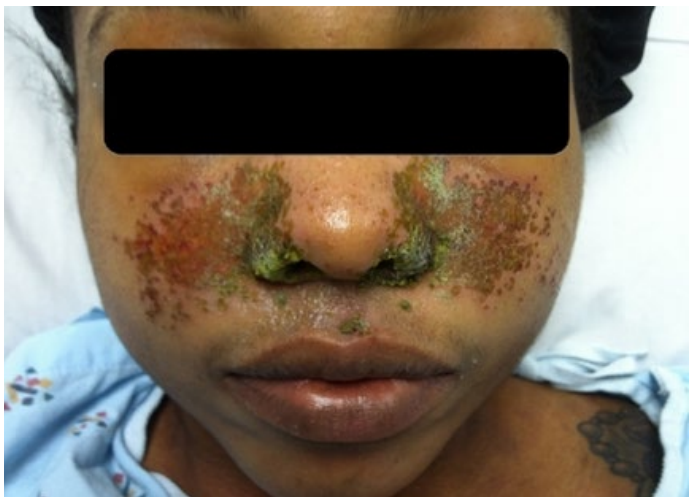
HSV–induced eczema herperticum is seen in clinical practice most commonly as lesions of the mucosa or epidermis.

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

Evaluation

Disseminated HSV may present with or without mucocutaneous lesions. When lesions are present, look for herpetic vesicles, pustules, and erosions. Constitutional symptoms often occur and commonly consist of fever, chills, myalgias, and regional lymphadenopathy.

Testing of plasma for HSV by PCR is the preferred diagnostic test in patients with disseminated HSV infection as viremia is always considered abnormal.



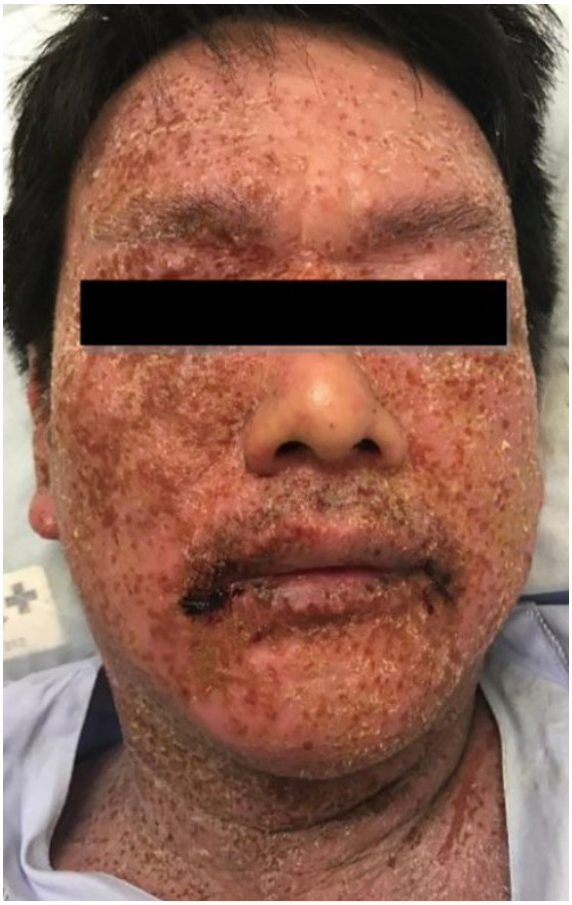
Management

Disseminated HSV with visceral disease is associated with high morbidity and mortality, so early recognition and prompt initiation of intravenous acyclovir (5 mg/kg every eight hours in patients with normal renal function) are critical. Transfer patients with suspected disseminated HSV to HLOC. Disseminated disease, often with prominent hepatitis, is important to consider in the differential diagnosis of pregnant people with hepatitis.[8] Please refer to the Sexually Transmitted Infection Care Guide for more information.[8]

ECZEMA HERPETICUM

Characteristics

Eczema herpeticum is a disseminated cutaneous infection caused by herpes simplex virus (HSV) and affects people with atopic dermatitis and other inflammatory skin diseases. Patients with eczema herpeticum are more likely to have food and environmental allergies, asthma, early onset of atopic dermatitis before age 5, and a history of *Staphylococcus aureus* and molluscum contagiosum infections.



Evaluation

Eczema herpeticum typically presents as a sudden onset eruption of monomorphic vesicles and "punched-out" erosions and ulcers with hemorrhagic crusts. These pruritic and painful lesions are superimposed on areas of preexisting atopic dermatitis, most commonly on the face, neck, and upper trunk. Eczema herpeticum lesions may spread to involve normal skin over 7-10 days. Lesions that are secondarily impetiginized may have an overlying honey-colored crust. Patients may have systemic symptoms like fever, lymphadenopathy, or malaise. Presentation ranges from mild and self-limiting in healthy adults to life-threatening in immunocompromised patients.

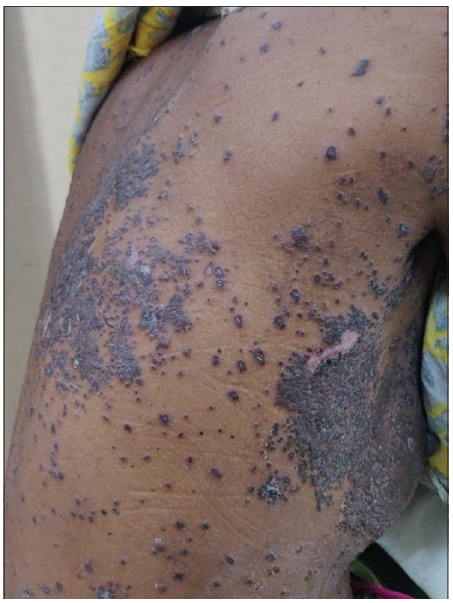
Viral polymerase chain reaction (PCR) can be performed on vesicle fluid to confirm the diagnosis and determine the type of herpesvirus with high sensitivity and specificity. Bacterial culture should be done if there is a concern for impetiginization.

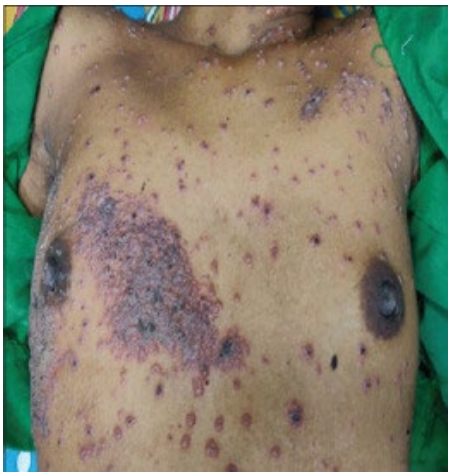
Management

Patients with eczema herpeticum should be treated promptly with systemic acyclovir or valacyclovir to minimize the risk of complications and prevent progression to severe disease.[8] Please refer to the Sexually Transmitted Infection Care Guide for more information.[8]

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

DISSEMINATED HERPES ZOSTER





Characteristics

Herpes zoster, also known as zoster or shingles, is caused by varicella zoster virus (VZV), the same virus that causes varicella (chickenpox). Primary infection with VZV causes varicella. Although zoster most often develops in persons with a history of varicella disease, the attenuated virus in the varicella vaccine can also establish latency and reactivate as zoster.

Patients who are immunocompromised may present with complicated zoster, which is defined as ocular, otic, neurologic, or disseminated infection. Disseminated herpes zoster (DHZ) includes extensive cutaneous involvement (i.e., >2 contiguous dermatomes, lesions involving both sides of the body or non-contiguous dermatomes), and/or visceral involvement.

Evaluation

The first visible symptom of zoster is a painful vesicular rash. Vesicles typically dry and crust in 7- 10 days, clearing completely within 2-4 weeks. In the 2-4 days prior to developing the rash, people often have pain, itching, or tingling in the location where the rash will appear. DHZ is more widespread and involves >2 dermatomes.

Management

Consider transfer of patients with DHZ to HLOC. In such patients, intravenous acyclovir (10 mg/kg every eight hours) should be initiated. When lesions are improving the patient can be transitioned to oral treatment such as valacyclovir (1 g three times daily). The duration of therapy is typically 10 to 14 days but may need to be extended in those with ongoing symptoms.

Prevention

Persons with zoster do not transmit zoster to others. However, they can transmit VZV, which can cause varicella in susceptible people. The infectious period begins when vesicles appear and ends when the vesicles have dried and developed crusts. A person is not infectious before vesicles appear.

Transmission of VZV from persons with zoster can occur via direct contact with fluid from zoster lesions or aerosolization of lesion material. There is also some evidence that VZV can be shed in the respiratory aerosols of persons with zoster. Persons with disseminated zoster are more infectious than persons with localized zoster.

Therefore, prevent the spread of VZV from patients with zoster by implementing the following appropriate infectious control precautions:

- Immunocompetent patients with localized zoster: Use standard precautions and completely cover lesions.
- Immunocompetent patients with disseminated zoster: Use standard precautions plus airborne and contact precautions until lesions are dry and crusted.
- Immunocompromised patients with localized zoster: Use standard precautions plus airborne and contact precautions until disseminated infection is ruled out, then use standard precautions until lesions are dry and crusted.
- Immunocompromised patients with disseminated zoster: Use standard precautions plus airborne and contact precautions until lesions are dry and crusted[9]

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

BURNS

Burns result from acute traumatic injuries to the skin or other tissues caused by heat with or without friction, electrical discharge, chemicals, or radiation. The traditional classification of burns as first, second, third, or fourth degree was replaced by a system reflecting the need for surgical intervention:

- **Superficial**, or epidermal, burns involve only the epidermal layer of skin and are painful, dry, red, and blanch with pressure.
- **Superficial partial thickness** form blisters within 24 hours between the epidermis and dermis. They are painful, red, weep, and blanch with pressure
- **Deep partial thickness** burns extend into the deeper dermis causing damage hair follicles and glandular tissue. They are painful to pressure only, almost always blister (easily unroofed), are wet or waxy dry, and have variable mottled colorization from patchy cheesy white to red
- **Full thickness** burns extend through and destroy all layers of the dermis and often injure the underlying subcutaneous tissue. Burn eschar, the dead and denatured dermis, is usually intact, which can compromise the viability of a limb or torso if circumferential. Burn areas that are waxy white or leathery gray and insensate characterize full-thickness burns.
- **Fourth-degree** burns are deep, potentially life-threatening injuries that extend through the skin into underlying soft tissue and can involve muscle and/or bone. There is no feeling in the area since the nerve endings are destroyed.

This section addresses the clinical assessment and management of different types of burns.

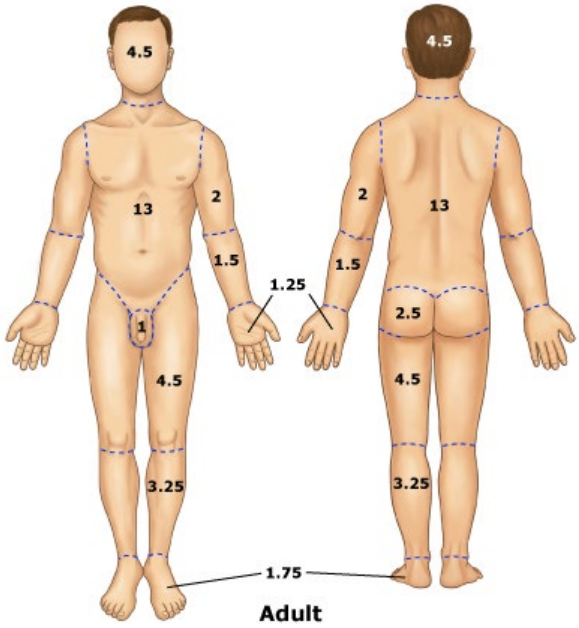
THERMAL BURNS

Characteristics

The most common thermal burns are associated with flames, hot liquids, hot solid objects, and steam. The depth of the thermal injury is related to contact temperature, duration of contact with the external heat source, and the thickness of the skin. Because the thermal conductivity of skin is low, most thermal burns involve the epidermis and part of the dermis.

Evaluation

Assess the environment for hazards to ensure it is safe and secure before attempting to reach the patient, then bring the patient to the clinical setting. A thorough and accurate estimation of burn size is essential to guide therapy and to determine when to transfer a patient to a burn center. Using one of the methods described below, the extent of burns is estimated and expressed as the total percentage of body surface area (TBSA). Superficial burns are **not** included in percentage TBSA burn assessment. Lund-Browder is the most accurate method for estimating TBSA, but the "Rule of Nines" is the most expeditious method to estimate TBSA

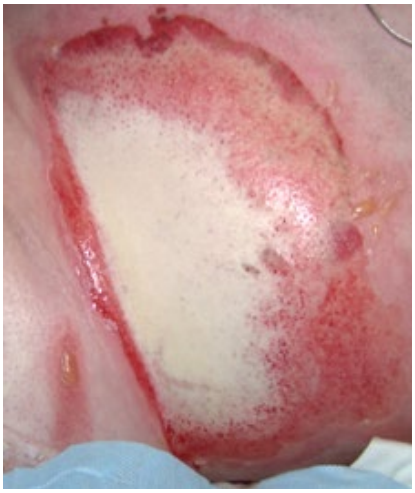


- Head represents 9% TBSA
- Each arm represents 9% TBSA
- Each leg represents 18% TBSA
- Anterior and posterior trunk each represent 18% TBSA

Superheated gases can cause burns to the respiratory system. More commonly, injury occurs from smoke exerting its effects through local chemical irritation. In addition, chemicals such as carbon monoxide or cyanide can lead to systemic toxicity. Direct thermal injury from hot smoke usually burns only the pharynx while steam can also burn the airway below the glottis. Assess patients with signs of potential inhalation such as facial flash burns, singed facial hairs, or smoke exposure.

Finally, assess for concomitant traumatic injuries by looking for friction burns, abrasions, fractures, hemorrhage, etc. Injury from friction can occur due to a combination of mechanical disruption of tissues and heat generated by friction.

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY



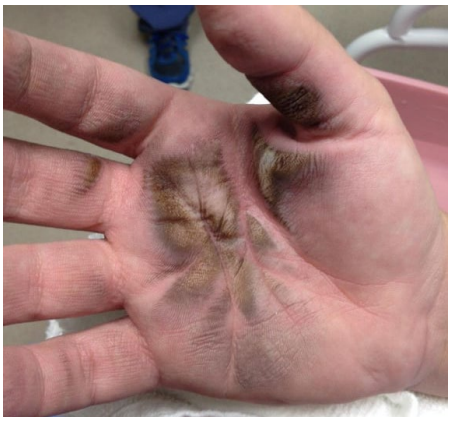
Management

Consider transfer to HLOC for

- All potentially deep burns of any size (i.e. deep partial thickness burns or deeper)
- Partial thickness burns ≥10% TBSA
- Burns on the face, hands, feet, genitalia, perineum, or over any joints
- Circumferential burns
- Patients with burns and concomitant traumatic injuries
- Patients with significant burns and other comorbidities, such as diabetes, hypertension, heart disease, kidney disease, etc.
- Patients with suspected inhalational injury

For small, localized superficial and superficial partial thickness burns, remove clothing, jewelry, and nonadherent debris. Cool the burns with room-temperature or cool running water or saline-soaked gauze. Clean wound with mild soap and water. Sloughed or necrotic skin, including ruptured blisters, should be debrided before applying a dressing. Ruptured blisters should be debrided (i.e., remove the entire blister and all loose skin so that no necrotic epidermis remains). Small intact blisters <2 cm in diameter do not need to be debrided. Superficial burns do not require dressings. Partial thickness burns are generally dressed in a topical agent two to three times per day. Superficial burns and superficial partial-thickness burns rarely develop such infections, so a topical antimicrobial agent is not required in these cases.[10,11]

ELECTRICAL BURNS



Characteristics

Electrical energy is transformed into heat as the current passes through poorly conducting body tissues. Electroporation (injury to cell membranes) disrupts membrane potential and function. The magnitude of the injury depends on the pathway of the current, the resistance to the current flow through the tissues, and the strength and duration of the current flow. Assess for entrance and exit burns.

Electrical burns account for 3-4% of all burn-related injuries, but approximately 40% of serious electrical injuries are fatal.

Evaluation

Assess the environment for hazards to ensure it is safe and secure before attempting to reach the patient, then bring the patient to the clinical setting. Electrical current can lead to ventricular dysrhythmia or respiratory arrest, so implement resuscitative efforts as indicated.

Superficial, partial-thickness, and full-thickness thermal burns can occur following electrical injury. Dry skin has a high resistance (approximately 100,000 ohms) and generates heat when exposed to a current, resulting in skin burns and potentially burns to adjacent tissue. However, dry skin is protective to internal organs because it prevents passage of current. The protective effects are lost with wet skin, which has a much lower resistance (less than 2500 ohms) and generates less heat but passes more current to internal organs for an equivalent voltage. Similar to thermal burns, assess the extent and depth of burn injury.

Management

Cardiopulmonary resuscitation may be needed. Do not stop resuscitation based on fixed, dilated, or asymmetric pupils in lightning victims since pupil function can be affected due to autonomic dysfunction instead of brain injury. Good outcomes have been reported even among patients with asystole. Clinical judgment should be used to determine the appropriate duration of resuscitative efforts.

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

Once stable, patient who have sustained electrical burns should be transferred to HLOC. Immobilize the cervical spine as appropriate, given the risk of secondary trauma. Evaluate for coexisting smoke inhalation or airway burns. Recognizing that surface findings may underestimate the extent of tissue damage or necrosis, should be used to guide management. Some injuries may not be apparent initially and become evident later in the patient's course.[10,11]

CHEMICAL BURNS

Characteristics

A wide range of caustic reactions, including alteration of pH, disruption of cellular membranes, and direct toxic effects on metabolic processes, causes injury. In addition to the duration of exposure, the nature of the agent will determine injury severity. Contact with acid causes coagulation necrosis of the tissue, while alkaline burns generate liquefaction necrosis. Systemic absorption of some chemicals is life-threatening, and local damage can include the full thickness of skin and underlying tissue.

Chemical burns are unique injuries that require individualized evaluation and management depending upon the causative agent. They are often occupational exposures and account for 4 percent of admissions to burn units.

Evaluation

Don personal protective equipment (PPE), include a gown, a mask, and goggles or a face shield. Assess the environment for hazards to ensure it is safe and secure before attempting to reach the patient, then bring the patient to the clinical setting. To ensure the safety of health care workers and others in the vicinity of the patient, remove all patient clothing and jewelry, then store in a secure container. Brush any dry chemicals with a dry brush or towel. Irrigate all wounds and any contaminated area with copious amounts of lukewarm water either with a shower or a hose at low pressure. If possible, determine the type of chemical.

The presence of dyspnea, cough, hoarseness, drooling, stridor, tachypnea, decreased breath sounds, wheezing, rales, rhonchi, or use of accessory respiratory muscles suggests a caustic chemical inhalation with upper airway or lung parenchymal edema or injury.

Assess for systemic toxicity since some chemicals are absorbed through the skin or produce vapors, fumes, or aerosols absorbed through the lungs. Similar to thermal burns, assess the extent and depth of burn injury.

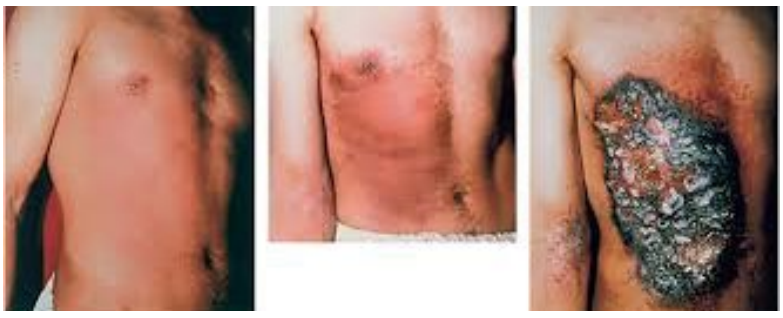
Management

Consider transfer to HLOC for all chemical burns and all patients with suspected inhalational injury.[10,11]

RADIATION BURNS

Characteristics

The most common type of radiation burn is sunburn. Radiation burns are most commonly seen today following therapeutic radiation therapy and are also seen in patients who receive excessive radiation from diagnostic procedure. Radiofrequency energy or ionizing radiation can cause damage to skin and tissues. The clinical results of ionizing radiation depend on the dose, time of exposure, and type of particle that determines the depth of exposure.



Radiation dermatitis is one of the most common side effects of radiotherapy for cancer, affecting over 90 percent of patients receiving radiotherapy. Cutaneous adverse effects of radiation therapy can be divided into early/acute reactions, occurring within 90 days of initiating treatment, and late effects, which often become apparent months to years after radiation treatment has been completed. Severe radiation burns, which cause full thickness skin necrosis and ulceration,

DERMATOLOGIC CONDITIONS: EMERGENCY DERMATOLOGY

may occur rarely as a result from high exposure to radiation during repeated diagnostic medical imaging, interventional radiology procedures, or radiation therapy.

Evaluation

The most distinctive feature of radiation burns compared with thermal burns is the difficulty in delineating radiation-injured tissue from uninjured tissue. This difficulty stems from the unpredictable progression of tissue injury, which can occur weeks to years after irradiation and often results in necrosis of skin grafts. Additional unique characteristics of radiation burns include a dose-dependent clinical pattern and opiate-resistant pain.

Management

Most radiation burns (e.g. sunburns) are mild and can be treated with cleansing and moisturizing the area to avoid secondary infection.

For patients on radiation therapy who develop mild grade 1 dermatitis, low-medium potency (group 4 and 5) topical corticosteroids may be used to control itch and irritation. For patients with more severe symptoms (grade 2 to 4 dermatitis), refer to specialists, like radiation oncology and Wound Central Team, for comanagement. Surgical intervention may also be necessary.[10,11]

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

BENIGN NEOPLAMS AND HYPERPLASIAS

Distinguishing common benign skin neoplasms and hyperplasias from potentially malignant tumors is important. Benign skin lesions can be diagnosed by their history, distribution, and morphology. A biopsy or excision is indicated if there is diagnostic uncertainty, if a lesion undergoes uncharacteristic or rapid change, or if a lesion causes pain or affects function.

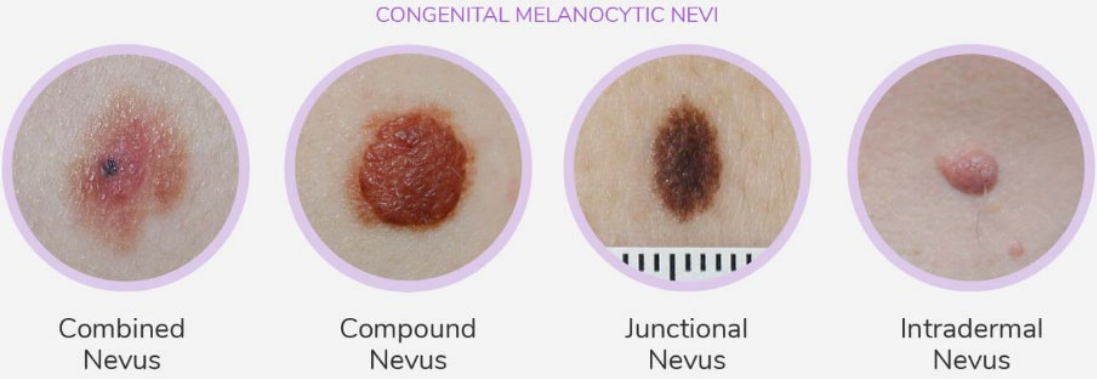
Primary Morphology → **Configuration** → **Demarcation** → **Color** → **Distribution** → **Secondary Morphology**

MELANOCYTIC (COMMON) NEVUS

Characteristics

A melanocytic nevus or mole is defined as benign neoplasms of melanocytes, most of which manifest themselves as cutaneous pigmented lesions. In general, the benign melanocytic nevus is a small (less than 6 mm), symmetrical, well-circumscribed proliferation of nested melanocytes. Melanocytic nevi are commonly divided into two major categories: congenital and acquired.

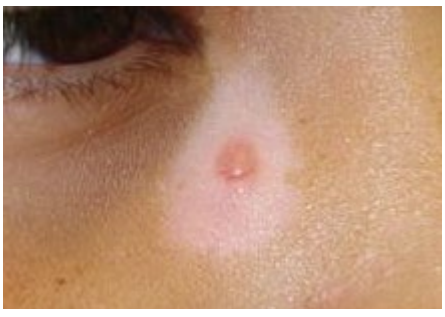
Congenital nevi are present at birth or appear within the first year of life and range from a few millimeters to sizes that may cover the majority of a body surface area (e.g. the garment nevus). They typically measure less than 6 mm and usually have one homogenous color, though the color may change over time.



It is normal for newly acquired nevi to arise at certain times, such as adolescence and pregnancy. Acquired nevi appear after the age of six months, reach a peak count in the third decade, and then slowly regress with age. It is also normal for pre-existing nevi to undergo normal, benign changes over time. However, patients with more than 100 melanocytic nevi have a 7-fold increase in risk for melanoma and should be monitored. In individuals with multiple nevi, the predominant group of nevi that share similar clinical and dermoscopic features defines the "signature nevus".

Management

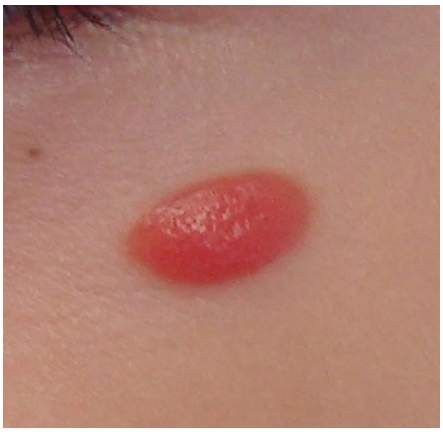
Most melanocytic nevi do not have to be biopsied and require no treatment other than longitudinal observation. Refer patients with multiple acquired nevi, patients with a halo nevus, to dermatology for routine follow-up with periodic full body skin examinations, and counsel patients on sun protection.



DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

Clinical indications to refer a patient to dermatology for a biopsy include:

- Lesions on the palms or soles (acral melanocytic nevi) with asymmetry, mottled pigmentation, and/or > 6 mm in diameter
- Single pigmented bands originating in the nail matrix that are dark in color or are ≥ 3 mm wide
- Nevi with new asymmetry based on border and/or color variation
- Nevi with new areas of pink, red, gray-blue, or white
- A new blue nevus
- A blue nevus that has superimposed changes (e.g., a papulonodule)
- A new halo nevus
- A halo nevus in which the central nevus surrounded by a depigmented patch has atypical or worrisome features
- A Spitz nevus with atypical clinical features (eg, diameter >1 cm, asymmetry, or ulceration)[12]

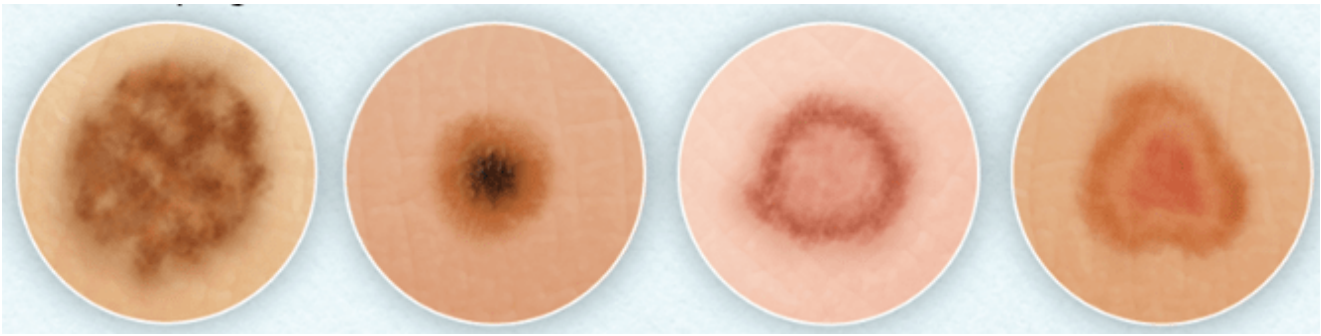


ATYPICAL (DYSPLASTIC) NEVUS

Characteristics

In contrast to a melanocytic nevus, an atypical nevus is defined as a benign neoplasm of melanocytes that display disorganized cells at least 5 mm in one dimension with a flat component and has at least two of following:

- Variable pigmentation
- Irregular asymmetric outline
- Indistinct border or unusual shape



Atypical nevi are assessed clinically, and those that show atypical architectural and cytologic features differing from common nevi are classified as dysplastic. It is important to note that in practice, the terms *dysplastic* and *atypical* are sometimes used as a modifier for other melanocytic neoplasias (e.g., Spitz nevi) or hyperplasias to indicate a variation from a typical pattern or to recognize an increased concern for malignant biologic potential.

Although atypical nevi are benign, the presence of even a single atypical nevus confers a high-risk of sporadic melanoma, increasing 10-fold with presence of 5 atypical nevi. Atypical nevus progression to melanoma rarely occurs and most often manifests as a new focal area of darker pigmentation or firm elevation. The rarity of such an event is supported by the observation that atypical nevi are far more common than melanoma and that more than one-half of the melanomas that occur in patients with atypical nevi develop de novo and not from a precursor atypical nevus. Moreover, studies of nevus-associated melanomas (i.e., melanomas that show nevus remnants on histopathology) indicate that they are associated with common nevi or atypical nevi with similar frequency.

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

Atypical nevi usually first appear during puberty and may develop throughout life. Atypical nevi arising on the scalp, breasts, or buttocks during childhood may be an early sign of high total body nevus count in adulthood. typical nevi are most often seen on the trunk and extremities, although they can develop anywhere on the body, including the scalp, breasts, buttocks, and genitalia. They rarely develop on the chronically sun-exposed skin of the face or hands.

In individuals with multiple nevi, the predominant group of nevi that share similar clinical and dermoscopic features defines the "signature nevus". The identification of nevi that appear different from the signature nevi, called "ugly ducklings" or "outliers," has been suggested as a screening method for the detection of melanoma in patients with many nevi, including those with many atypical nevi.

Management

The diagnosis of atypical nevus is usually clinical, based upon the finding of a predominantly macular, pigmented lesion that is larger than common melanocytic nevi (>5 mm), with an irregular shape, ill-defined border, and variable color with areas of pink, tan, brown, or dark brown. Other colors, such as blue, red, or white, are not usually seen in atypical nevi, and their presence increases the probability that the lesion is a melanoma. Only a dermatopathologist can distinguish atypical nevi from melanoma, so refer patients with an atypical nevus to dermatology for close follow up with dermoscopy and/or definitive diagnosis with biopsy.

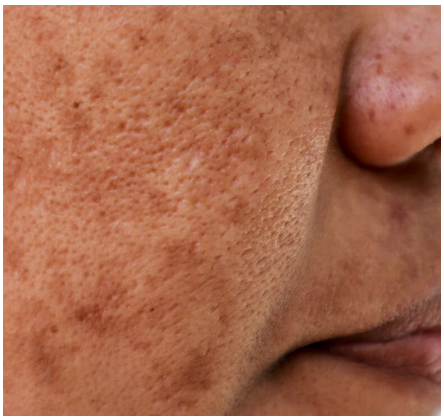
Since atypical nevi are benign lesion, most do not require surgical removal. However, risk assessment, prevention, and early detection of melanoma are key aspects of the lifelong management of patients with an atypical nevus. Follow-up of these patients include:

- Assessing the patient's personal and family history of melanoma
- Total body skin examination
- Excision and histologic examination of suspicious lesions by a dermatopathologist
- Educational on skin self-examination and sun protection
- Routine ophthalmic examination for ocular nevi and/or ocular melanoma

Refer patients with new, changing, or symptomatic lesions that are suspicious of melanoma to dermatology for excision then histologic examination by a dermatopathologist.[12]

LENTIGO

Characteristics



Lentigines are benign pigmented macules that result from increased activity of epidermal melanocytes. In contrast to ephelides (freckles) that are often seen in children with lightly pigmented skin and fade in the absence of sun exposure, lentigines are persistent. There are two major types of lentigines: simple lentigo and solar lentigo.

Simple lentigines often appear during childhood as sharply circumscribed, round-to-oval, uniformly brown or brownish-black macules that are usually <5 mm in diameter. There are typically few lesions, with no predilection for sun-exposed sites.

In contrast, solar lentigines appear only in sun-exposed areas, particularly in sites of greatest cumulative exposure (eg, the face, dorsal hands, extensor forearms, upper trunk). Because the incidence of solar lentigines increases with sun exposure, they are most often seen in adults. Multiple tan to dark brown macules are typically present. The lesions range from a few millimeters to >1 cm in diameter. Confluent solar lentigines often merge to form larger patches that can be irregular in shape. These changes, known as photoaging, are caused by repeated sun exposure and not by the passage of time. Many of the clinical signs attributed to aging are manifestations of solar damage.

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

Management

Although solar lentigines themselves have no malignant potential, they represent a sign of photodamage that indicates an increased risk for the development of melanoma and nonmelanoma skin cancers during adulthood. In young adults with solar lentigines, consider doing an annual skin examination. If the patient also has atypical or numerous nevi, or a family history of melanoma, then refer to dermatology.

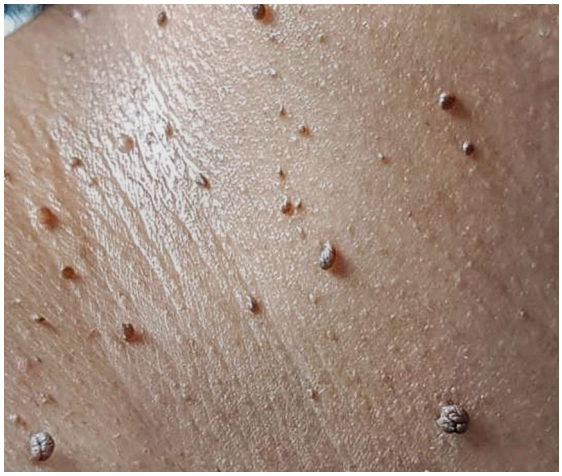
Since confluent solar lentigines form patches of irregular shape, this can often be difficult to distinguish from melanoma. Consider lentigo maligna for lesions that are irregular in shape, color, and size, and refer to dermatology.

Triple combination therapy (fluocinolone 0.01%/hydroquinone 4%/tretinoin 0.05%) can enhance resolution of solar lentigines treated with cryotherapy.

ACROCHORDONS

Characteristics

Acrochordons, commonly known as skin tags, are benign outgrowths of normal skin causing a dermal tumor. They usually appear as skin-colored pedunculated lesions on a narrow stalk. Acrochordons usually occur in sites of friction, particularly the eyelids, axilla, neck, inframammary, and inguinal regions. Risk factors for the development of acrochordons are:



- Age
- Overweight and obesity
- Diabetes mellitus
- Metabolic syndrome
- Second trimester of pregnancy

Management

Most acrochordons are asymptomatic. Treatment is indicated for acrochordons that are:

- Frequently traumatized from jewelry, clothing, shaving, or itching
- Develops over the eyelids and affects vision
- Infarcted, which occurs from torsion of the peduncle

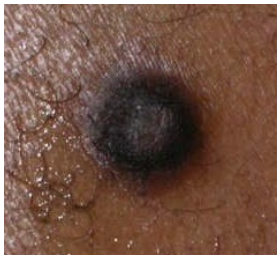
Consider applying ethyl chloride spray for local anesthesia prior to removal. 1% lidocaine-epinephrine may be required for larger lesions to lessen bleeding and lower discomfort upon removal. Treatment options include:

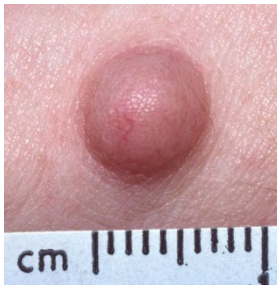
- Cryosurgery with liquid nitrogen
- Snip excision with forceps and fine-grade scissors
- Shave excision
- For larger lesions, which often bleed freely, consider refer to dermatology for electrosurgery/electrodesiccation

Lesions are unlikely to recur after removal, but new lesions develop in predisposed skin areas.[14]

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

DERMATOFIBROMA





Characteristics

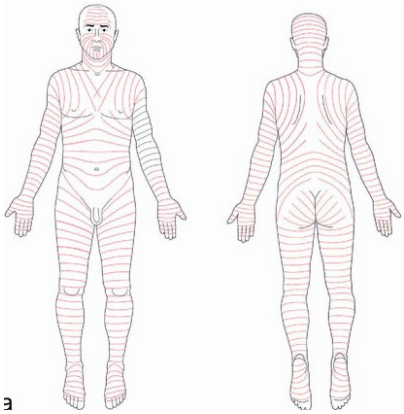
Dermatofibroma, also known as fibrous histiocytoma, is a common, benign, cutaneous soft-tissue lesion characterized by firm subcutaneous nodules, typically measuring 1 cm or less in diameter. Dermatofibromas are commonly found on the extremities and are prevalent across all age groups but are more commonly found among women ages 20-50 yo.

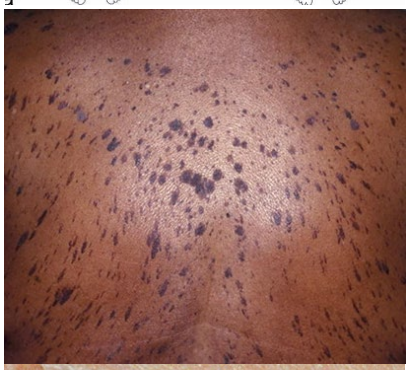
Management

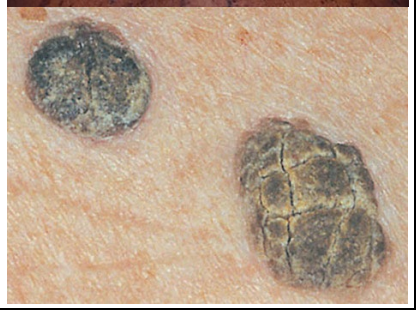
Most dermatofibromas are asymptomatic but may sometimes cause pain, tenderness, or itchiness. Treatment is indicated for dermatofibromas that cause symptoms.

Excision for histopathological examination is indicated when the lesion shows signs of malignancy, such as changes in appearance or bleeding. With thorough excision, these lesions rarely recur, and only the most aggressive variants show local recurrence. Complications associated with dermatofibroma primarily arise from surgical removal and may include bleeding, infection, scarring or disfigurement, and the necessity for additional procedures.[14]

SEBORRHEIC KERATOSIS







Characteristics

Seborrheic keratosis, the most common benign cutaneous neoplasms, are warty, age-related hyperkeratotic papules and plaques that appear anywhere on the body, most commonly the trunk. Seborrheic keratosis does not form on the palms and soles. Seborrheic keratosis often follow the pattern of Langer's lines, meaning they tend to appear along the direction of skin tension lines, particularly on the trunk where they can be seen in a linear distribution following the cleavage lines of the body. This is most noticeable when multiple seborrheic keratoses develop in a clustered pattern on the torso.

Unlike melanoma which appear and progress quickly, seborrheic keratosis is caused by the benign proliferation of immature keratinocytes, resulting in well-demarcated, round or oval, flat-shaped macules. They are typically slow-growing, can increase in thickness over time, and they rarely resolve spontaneously seborrheic keratosis lesions will have a long development period and a minimal progression. The warty surface usually has a waxy, stuck-on-the-skin look. The color of seborrheic keratoses is usually brown but can range in color from white to black. Seborrheic keratoses are painless, but some patients may complain of itching.

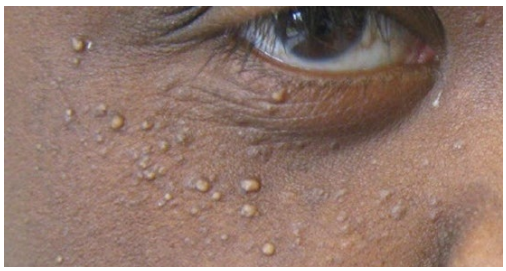
Seborrheic keratosis is typically seen in patients greater than 50 years of age and become more frequent as one ages. Although seborrheic keratosis lesions are more common in the middle-aged and elderly, they can also present in young adults. There is no prevalence difference between males and females. However, seborrheic keratosis appears to be more frequent in populations with lighter skin tones.

Management

No treatment is necessary. If the plaques are pruritic, they can be removed by curettage or cryotherapy.[14, 15]

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

MILIA



Characteristics

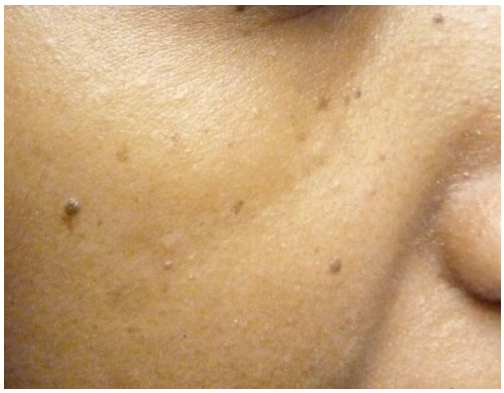
Milia are benign and transient subepidermal keratin cysts that can develop in any area of the skin, most commonly the face. They present as firm, skin-colored papules, 1 to 2 mm in diameter. Milia may develop spontaneously or as secondary lesions during the healing process of second-degree burns, blistering diseases (eg, epidermolysis bullosa, porphyria cutanea tarda), dermabrasion, and ablative laser resurfacing. They may also be an adverse effect of tyrosine kinase inhibitors.

Other benign cysts include epidermoid cysts, sebaceous cysts, pilar cysts, ganglion cysts, dermoid cysts, and pilonidal cysts.

Management

Milia can be treated for cosmetic reasons by incision of the overlying epidermis and expression of the content. Smaller lesions may respond to topical retinoids applied daily for several weeks. Recurrence is uncommon.[14]

DERMATOSIS PAPULOSA NIGRA



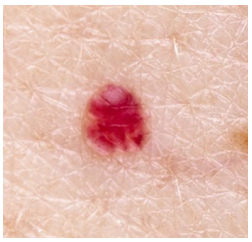
Characteristics

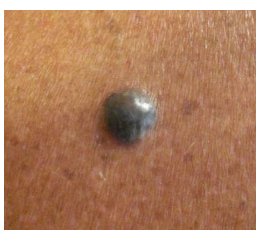
Dermatosis papulosa nigra is a common benign skin condition that occurs predominantly in dark-skinned persons with strong genetic potential. It presents as 1- to 5-mm lesions appearing as multiple brown to dark-brown, dome-shaped papules on the face, neck, and trunk. The face is the most common location, with the malar and temple areas often involved. Onset is typically after puberty, and lesions increase in number over time. Usually there are no associated symptoms, although the lesions can occasionally be pruritic or become irritated.

Management

Treatment is not required, but cosmetic therapeutic options include scissor excision, laser therapy, electrodessication, curettage, cryotherapy, and microdermabrasion. Caution should be used in selecting a treatment modality because of the increased risk of post inflammatory hypo- or hyperpigmentation in persons with darker skin.[15]

CHERRY ANGIOMA





Characteristics

Cherry angiomas, also known as Campbell de Morgan spots, are mature capillary proliferations that are common in middle-aged and older adult patients. They tend to be darker red or purple in individuals with darkly pigmented skin. They usually occur as multiple lesions, most commonly on the trunk, and bleed profusely with any traumatic rupture.

Other benign vascular tumors include hemangioma, pyogenic granuloma, and spider angioma.

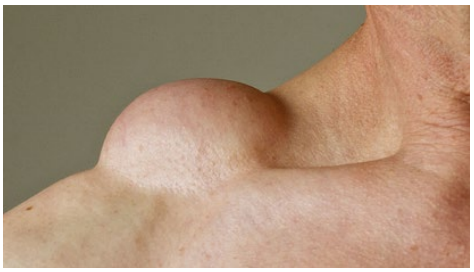
Management

If there is any concern for malignancy, the lesion should be excised and sent for histopathologic examination.

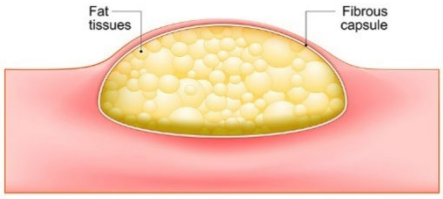
Treatment of benign vascular tumors is only necessary for patients who are bothered by the lesions.[14]

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

LIPOMA AND ANGIOLIPOMA



Lipoma



Characteristics

Superficial subcutaneous lipomas are the most common benign soft-tissue neoplasms. They consist of mature fat cells enclosed by thin fibrous capsules. Lipomas can occur on any part of the body and usually develop superficially in the subcutaneous tissue. Rarely, they may involve fascia or deeper muscular planes. Lipomas present as soft, painless subcutaneous nodules ranging in size from 1 to >10 cm. They occur most frequently on the trunk and upper extremities and can be round, oval, or multilobulated. Frequently, patients may have more than one lipoma.

Angiolipomas closely resemble lipomas but are usually painful and tender and frequently occur in multiple. Angiolipoma appears as a small, well-circumscribed, subcutaneous tumor.

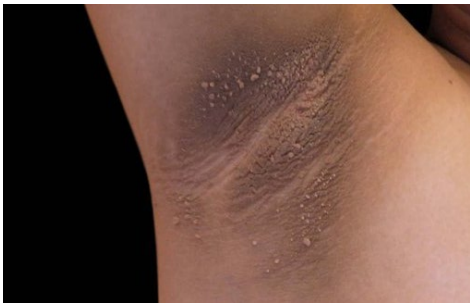
Malignant transformation is rare.

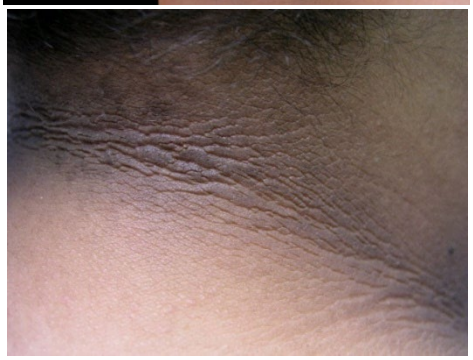
Management

Ultrasound examination can be helpful to distinguish a lipoma or an angiolipoma from an epidermoid cyst or a ganglion cyst. If a suspected lipoma or angiolipoma causes symptoms of pain or restriction of movement, is rapidly enlarging, or is firm rather than soft, a biopsy is indicated to confirm the diagnosis and exclude malignancy.

The treatment of lipomas, if needed because of pain, cosmesis, or concerns over diagnosis, is surgical removal of the fat cells and fibrous capsule. Recurrence of an excised lipoma is not common.[14]

ACANTHOSIS NIGRICANS





Characteristics

Acanthosis nigricans is a common condition that is characterized by smooth, velvet-like, hyperkeratotic plaques in intertriginous areas (e.g., groin, axillae, neck) due to epidermal and dermal hyperplasia. The thickened skin texture can feel leathery, and acrochordons (skin tags) can develop in affected areas. Typically, patients deny bothersome symptoms, unless acrochordons become irritated due to friction.

Three types of acanthosis nigricans have been recognized:

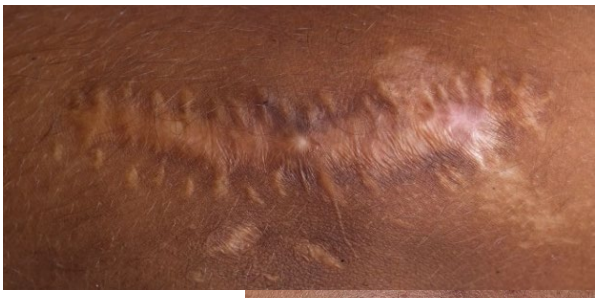
- Type I is associated with malignancy. Occasionally, acanthosis nigricans is a marker of an underlying adenocarcinoma, especially of the gastrointestinal tract (60% gastric). Malignant acanthosis nigricans has a sudden onset and more extensive distribution, including the face, palms, and trunk.
- Type II is the familial type, with autosomal dominant transmission. It is very rare and appears at birth or soon after. Type II has no malignancy association.
- Type III acanthosis nigricans is associated with obesity and insulin resistance. Type III is the most commonly occurring type. Acanthosis nigricans can develop following the use of some medications, such as systemic corticosteroids, nicotinic acid, diethylstilbestrol, and isoniazid (INH).

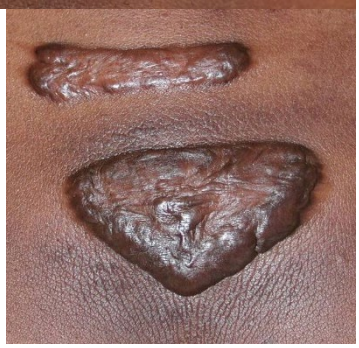
Management

Treat the underlying cause of acanthosis nigricans.[17]

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

KELOIDS AND HYPERTROPHIC SCARS





Characteristics

Keloids are benign growths that represent an overgrowth of scar tissue at sites of trauma, such as acne, burns, surgery, ear piercing, tattoos, and infections. Keloids are smooth, shiny, and firm papules, plaques, and nodules. They tend to be reddish-pink to dusky violet with progressive hyperpigmentation. Common locations are ear lobes, jaw line, posterior neck, scalp, chest, and back. Lesions are sometimes asymptomatic, but often are associated with pruritus, pain, and hypersensitivity. There is a higher incidence in Black, Latino/Hispanic, and Asian patients. The most common ages at presentation are 10-30 yo.

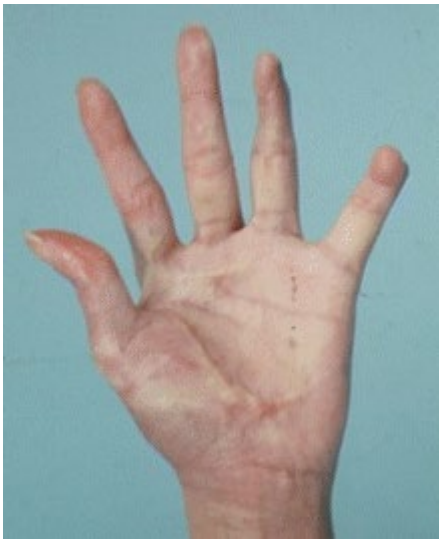
Keloids are often confused with hypertrophic scars. Hypertrophic scars typically develop soon after the inciting trauma and are found on areas of the body with frequent motion, such as extensor surfaces. They are confined to the borders of initial injury and may regress. Conversely, keloids develop months to years after injury and are not usually located in areas of motion. Keloids extend beyond the borders of original injury, progressing over time.

Management

Patients with keloids often seek treatment for associated symptoms and cosmetic reasons. Early treatment offers the best outcome and many options are available. Often, multiple therapies are used in combination. A meta-analysis of several options, including intralesional steroids, interferon, fluorouracil, bleomycin, surgical excision, laser therapy, radiation, and cryotherapy, showed a 70% chance of improvement with treatment. Intralesional steroid injections are first-line therapy. Triamcinolone acetonide at lower concentrations (10 mg per mL) may be used initially and titrated up based on response. In general, higher concentrations can be used safely in firmer, more elevated lesions. Discuss possible adverse effects of atrophy, hypopigmentation, striae, and telangiectasias with the patient before therapy, and monitor for these adverse effects before each treatment. Lesions may be injected every 4-8 weeks. Surgical excision alone has a high recurrence rate.

Guidelines for the prevention of keloids and hypertrophic scars include avoiding excessive movements, friction, or scratching that stretch the wound after undergoing surgery or other invasive procedures and keeping wounds clean. Counsel patients prone to keloids to avoid unnecessary trauma to the skin, such as tattoos and piercings.[18]

CONTRACTURE SCARS



Characteristics

Contractures are an abnormal occurrence that happens when a large area of skin is damaged and lost, often times with burn injuries, resulting in a scar. The scar formation pulls the edges of the skin together, causing a tight area of skin. This can also occur as scars heal. The decrease in the size of the skin can then affect the muscles, joints, and tendons, causing a decrease in movement. Despite appropriate initial treatment and compressive therapy, contractures are common after deep burn. The most common and functionally limiting are web space and hand contractures. The classic burn claw hand deformity includes extension contracture of the metacarpophalangeal joints and flexion contractures of the proximal interphalangeal joints.

Management

For patients who have contracture scars that limit movement despite physical and occupational therapy, consider referring to plastic surgery or hand surgery, if indicated.[18]

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

PRECANCEROUS LESIONS

Precancerous lesions develop on skin that has been exposed to excessive and chronic ultraviolet (UV) radiation. Treatment for these precancerous lesions mentioned in this section can prevent progression to skin cancer and relive symptoms of itching and tenderness.

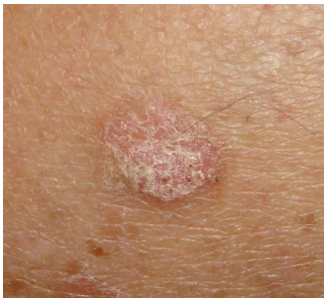
ACTINIC KERATOSIS

Characteristics

Actinic keratosis (AK), also known as solar keratosis, is a premalignant cutaneous lesion that may progress to cutaneous squamous cell carcinoma (cSCC). Often associated with chronic sun exposure, individuals with AKs may present with irregular, scaly papules or plaques on sun-exposed regions of the body. Common locations for AKs include the face, scalp among patients who are bald or have thinning hair, back of the arms, and dorsal aspect of the hands. AK is frequently diagnosed clinically through a combination of touch and visual inspection. Some macular lesions lack erythema or may be brown/hyperpigmented on darker skin and are more easily identified through the detection of rough texture.

The development of actinic keratosis is influenced by various independent risk factors, which may include:

- Age
- Male gender
- Pigmentary phototypes I and II
- Geographic locations closer to the equator
- Immunosuppression
- Excessive and chronic sun exposure
- History of prior skin cancer



Management

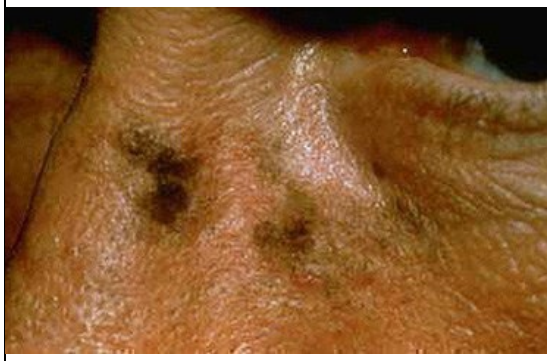
A biopsy is not routinely performed for the diagnosis of AK. However, a biopsy is indicated if the diagnosis is uncertain and to rule out cSCC. Indications for biopsy are:

- Lesions greater than 1 cm in diameter
- Lesions with underlying substance or induration
- Rapidly growing lesions
- Ulcerated lesions
- Tender lesions
- Lesions that fail to respond to appropriate therapy

There are multiple effective treatment options for AKs, including destructive therapies (e.g., cryosurgery, surgery, dermabrasion, photodynamic therapy [PDT]), topical medications (e.g., topical fluorouracil, imiquimod, diclofenac), and field ablation treatments (e.g., chemical peels, laser resurfacing). The choice of therapy is influenced by several factors, including:

- Number and distribution of lesions
- Lesion characteristics
- Patient preference
- Patient tolerance for side effects
- Treatment availability

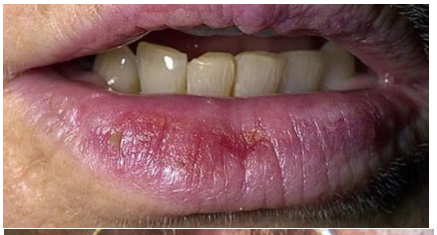
DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

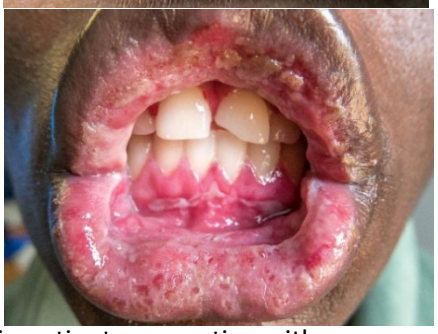


Consider comanagement of AKs with dermatology for suspected malignancy and for guidance of the most appropriate treatment. Reinforcing the use of sun protection measures, including wearing protective clothing and regular use of broad-spectrum sunscreens, is of key importance in the ongoing management of patients with AKs.

Ongoing monitoring for lesion recurrence and cutaneous malignancies at 6 to 12 months post-treatment is required for all patients with a history of AKs. Treatment with topical fluorouracil or imiquimod may be effective for the long-term control of AKs and prevention of cSCC.[19]

ACTINIC CHEILITIS





Characteristics

Similar to actinic keratosis, actinic cheilitis, also called solar cheilitis, is a premalignant disorder of the lip caused by chronic sun exposure. Given cutaneous squamous cell carcinoma (cSCC) on the lips is considered a high-risk form of skin cancer with an 11% chance of metastasis compared to 1% for other body locations, it is essential to recognize and appropriately manage these premalignant lesions.

Actinic cheilitis initially presents as a persistent area of dryness and desquamation, typically located on the lower lip along the vermillion border, which is likely to receive more ultraviolet radiation exposure than the upper lip. On palpation, these lesions feel rough, like sandpaper. Atrophic changes, blurred demarcation of the vermilion border, erythema, edema, hyperkeratotic (leukoplakia-like) plaques, ulceration, and crusting may be seen in more advanced lesions.

Management

A lip biopsy is not routinely performed in patients with a history of chronic sun exposure and early, obvious changes of actinic cheilitis. However, a biopsy is warranted in patients presenting with hyperkeratotic (leukoplakia-like) or nodular areas, with or without erosions or ulceration, that are suspicious for cSCC.

Treatments that are commonly used include topical medications (e.g., topical fluorouracil, imiquimod), destructive therapies (e.g., liquid nitrogen, electrodessication, chemical peels, laser therapy, photodynamic therapy), and surgery.

Consider comanagement of actinic cheilitis with dentistry and dermatology for suspected malignancy and for guidance of the most appropriate treatment. As patients with actinic cheilitis have an increased risk of developing lip cancer, recommend regular surveillance and repeated biopsies of suspicious areas. Reinforcing the use of sun protection measures, including wearing a hat or cap and using broad-spectrum sunscreen multiple times per day, is of key importance in the ongoing management of patients with actinic cheilitis. Cessation of tobacco use, both smoked and smokeless, should also be encouraged.[20]

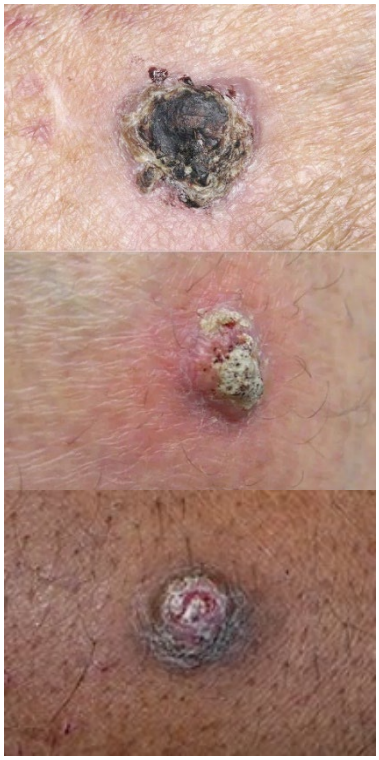
DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

SKIN CANCER

Skin cancer, including melanoma, basal cell carcinoma, and cutaneous squamous cell carcinoma, has one of the highest global incidences of any form of cancer. Since the main risk factor for all skin cancer is exposure to ultraviolet (UV) radiation, more than 80% of skin cancers are considered preventable. Providers play a vital role to tailor patient education about the risks of UV radiation and to detect lesions suspected to be skin cancer, since timely diagnosis and treatment can improve patient outcomes, particularly for melanoma.

However, when skin cancer develops in people of color, it is often in a late state when diagnosed because skin cancer can present differently on skin of color. Skin cancer often develops on parts of the body that get less sun, like the bottom of the foot, lower leg, and palms. Skin cancer may also begin under a nail, around the anus, or on the genitals.

CUTANEOUS SQUAMOUS CELL CARCINOMA



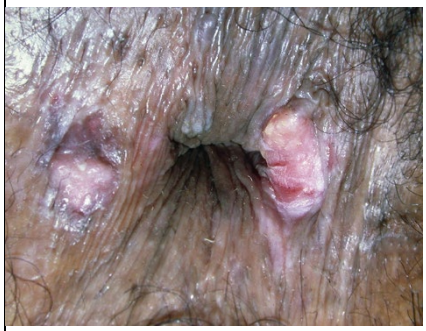
Characteristics

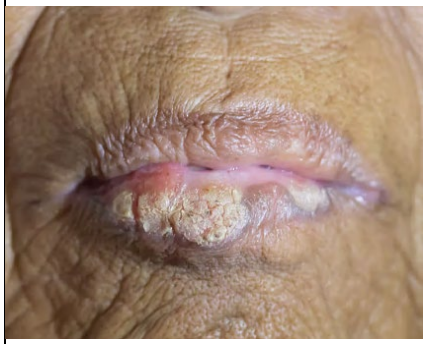
Cutaneous squamous cell carcinoma (cSCC) is a malignant tumor arising from epidermal keratinocytes. It is the second most common skin malignancy in the United States, and its incidence steadily rises annually so timely surveillance, early diagnosis, and prompt treatment are critical to minimize morbidity and mortality risks.

Risk factors include immunosuppression, chronic wounds, fair skin, male gender, older age, several genetic syndromes, environmental exposures such as ultraviolet radiation, and a previous history of cSCC. Although metastasis is rare, the most common site of metastasis is the lymph nodes.

In individuals with lightly pigmented skin, cSCC typically develops in areas of photodamaged skin and presents with a wide variety of cutaneous lesions, including papules, plaques, or nodules, that can be smooth, hyperkeratotic, or ulcerated. It can also present as a wart-like growth or an open lesion that does not heal.

Non-sun-exposed areas represent the most common location for cSCC in individuals with darkly pigmented skin. In Black patients, common sites for cSCC include the lower legs, anogenital region, and areas of chronic inflammation or scarring. Lesions that develop in relation to chronic scarring processes account for 20-40% of cSCCs in Black patients.





Genital and periungual cSCC lesions are less common and are usually related to infection with high-risk human papillomavirus (HPV). Of note, tumors arising on the ear, preauricular surfaces, or at mucocutaneous interfaces (i.e., lips, genitalia, and perianal area) tend to be more aggressive, with rates of metastasis from 10-30%.

Patients at risk for developing cSCC are those who have had excessive sun exposure, are immunocompromised, have had prior skin cancers, or occupational exposures to chemicals like arsenic and tar.

Management

Refer patients with suspected or confirmed cSCC to dermatology.

Skin biopsy is required to confirm the diagnosis and to provide information needed for staging. Sentinel lymph node biopsy and/or radiological evaluation for lymph node metastasis with computed tomography or ultrasound may be recommended.

The preferred therapeutic intervention for cSCC is surgical excision. Mohs micrographic surgery is preferred in cases that meet appropriate use criteria (AUC). Factors listed in the AUC include, but are not limited to, clinical diameter of the apparent lesion greater than 2 cm, high-risk histological features, recurrent versus primary lesions, cosmetically sensitive and/or high-risk anatomical locations such as the ears, lips, nose, and periocular regions, as well as immunosuppression.

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

For patients who are not suitable for surgery, options for treating cSCC include superficial radiation therapy, 5-fluorouracil cream, imiquimod cream, cryotherapy, photodynamic therapy, and/or ablative laser. However, these treatments often result in higher recurrence rates and lack histologically confirmed clearance.

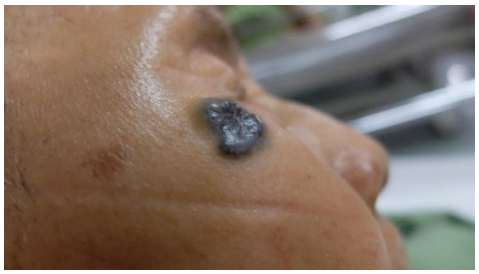
Referral to radiation or surgical oncology, urology, gynecology, or otolaryngology may be necessary for comprehensive management in advanced cases. Lymphadenectomy of the associated n⁻dal basin is recommended for cases with positive lymph nodes. Radiation therapy is typically used for cases involving large-caliber nerve invasion, lymph vascular invasion, multiple lymph nodes, or extracapsular extension. Adjuvant systemic oncologic therapy for advanced cases may include chemotherapy, epidermal growth factor inhibitors, or immunotherapy. Novel immunotherapeutic agents have shown superior clinical outcomes compared to traditional systemic therapies.

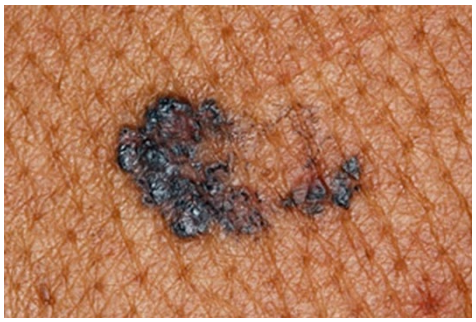
The American Academy of Dermatology (AAD) recommends the following preventative measures for patients:

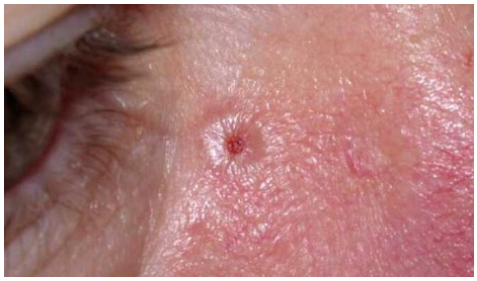
- Individuals should limit sun exposure during peak UV index hours.
- Sunscreen with at least SPF 30 should be regularly used and reapplied every 2 hours.
- Reapply sunscreen more frequently with excessive sweating.
- Wear protective clothing, a hat or cap , and sunglasses when exposed to sunlight.
- Do not use tobacco products.
- Self-skin check is recommended for patients to do at least once a month. This includes looking for any changes where the cancer was treated, as well as looking for any new areas of concern.
- Full-body skin exams are recommended every 3-6 months for the first few years, followed by longer time intervals up to 12 months between visits.[21]

BASAL CELL CARCINOMA

Characteristics







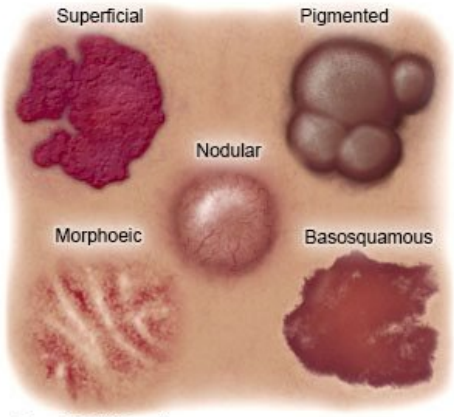
Basal cell carcinoma (BCC) a common cutaneous malignancy that occurs mostly on sun-damaged skin of the head, neck, and trunk. It is the most common cutaneous malignancy, affecting close to 1:5 Americans, and accounts for 80% of all non-melanoma-type skin cancer. BCC rarely metastasizes but can cause significant local destruction and morbidity if not recognized and adequately treated.
BCC develops at sites of prior ultraviolet light exposure. The latency period extends from 10-40 years, and no known precursor lesion exists. Individuals with light skin and lighter-colored eyes are at the greatest risk of developing these tumors. Longer life spans and an increase in the use of immunosuppressant medications has further added to the incidence of patients with basal cell carcinoma.

Basal cell carcinoma can be broken into three main categories: *nodular*, *superficial*, and *infiltrative*. Nodular basal cell carcinoma is the most common subtype and presents as a pink-to-violet, pearlescent papule with overlying telangiectasias and rolled borders. It commonly occurs on the head and neck, and it is a slow-growing papule that will often ulcerate and bleed. The latter may mislead patients to dismiss the neoplasm as a cut while shaving. Superficial basal cell carcinoma presents as a thin, pink plaque, papule, or macule with a pink, pearly border that is most commonly seen on the chest, back, or extremities. Infiltrative basal cell carcinomas include infiltrative, micronodular, and morpheaform subtypes. Morpheaform basal cell carcinoma presents as a firm, scar-like plaque and should be considered in the presentation of a new scar without previous trauma to the area. Infiltrative and micronodular basal cell carcinoma present similarly to nodular basal cell carcinoma as pink, pearly papules with overlying telangiectasias.

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

Management

Refer patients with suspected or confirmed BCC to dermatology.
A skin biopsy is required to confirm the diagnosis, as well as to provide information that is needed for staging and management. Additionally, sentinel lymph node biopsy and/or radiological evaluation for lymph node metastasis with computed tomography or ultrasound may be recommended.



Basal Cell Carcinoma

The preferred therapeutic intervention for BCC is surgical excision. Mohs micrographic surgery (MMC) is preferred in cases that meet appropriate use criteria (AUC). Factors listed in the AUC include, but are not limited to, clinical diameter of the apparent lesion greater than 2 cm, high-risk histological features, recurrent versus primary lesions, cosmetically sensitive and/or high-risk anatomical locations such as the ears, lips, nose, and periocular regions, as well as immunosuppression.

Chemotherapy creams, including imiquimod 5% cream and 5-fluorouracil 5% cream, are FDA approved to treat superficial basal cell carcinoma. Treatment regimens vary, but on average, both creams require application over multiple weeks. A major benefit of this approach over surgical intervention is that they can be used in cosmetically sensitive areas as they are less likely to cause scarring. Vismodegib was approved by the Food and Drug Administration (FDA) in 2012 for locally advanced and metastatic lesions of basal cell carcinoma.

Radiation therapy is a curative, standard treatment option in cases where surgery is not feasible or not preferred. It can also be used adjunctively in high-risk postoperative settings (e.g., with positive margins or extensive perineural invasion). In non-curative cases, it can be offered as palliation for local symptoms such as pain, ulceration or bleeding.

The prognosis for patients with BCC is good, demonstrating a 100% survival rate in cases that have not metastasized. However, if BCC goes untreated and progresses, the result can be significant morbidity with significant disfigurement.

The American Academy of Dermatology (AAD) recommends the following preventative measures for patients:

- Individuals should limit sun exposure during peak UV index hours.
- Sunscreen with at least SPF 30 should be regularly used and reapplied every 2 hours.
- Reapply sunscreen more frequently with excessive sweating.
- Wear protective clothing, a hat or cap, and sunglasses when exposed to sunlight.
- Do not use tobacco products.
- Self-skin check is recommended for patients to do at least once a month. This includes looking for any changes where the cancer was treated, as well as looking for any new areas of concern.
- Full-body skin exams are recommended every 6-12 months[22]

MALIGNANT MELANOMA

Characteristics

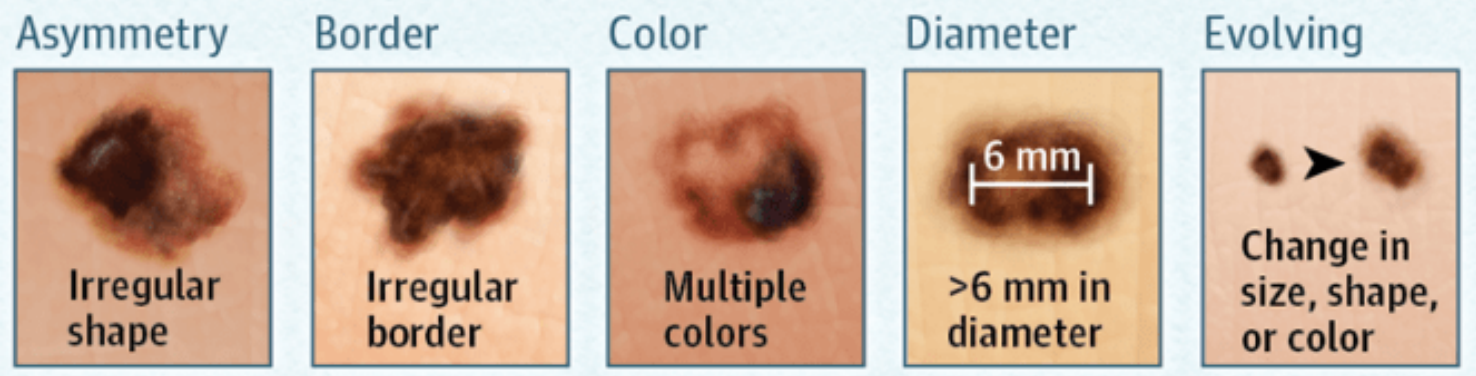
Melanoma is a malignancy derived from the malignant transformation of melanocytes. Melanocytes are derived from the neural crest. Therefore, melanomas, although they arise from the skin, can develop in other locations where neural crest cells migrate, such as the gastrointestinal tract and brain.

The incidence of primary cutaneous melanoma has increased steadily for several decades and remains the most lethal form of cutaneous neoplasm. The 5-year relative survival rate for patients with stage 0 melanoma or melanoma-in-situ is 97%, compared to 30% for those with stage IV disease. Mucosal and ocular melanomas typically have worse prognoses.
A full-body skin examination is the most essential and fundamental evaluation needed in diagnosing melanoma. The

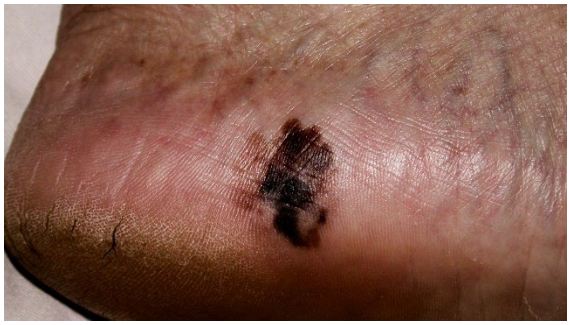
DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

characteristic signs of early melanoma are recognized with the following well-known ABCDE mnemonic:

- "**A**" stands for **A**symmetry
- "**B**" stands for **B**order: irregular, ragged, notched, or blurred edges
- "**C**" stands for **C**olor: nonuniform or variegated
- "**D**" stands for **D**iameter: larger than 6 millimeters
- "**E**" stands for **E**volving**:** changes in size, shape, or color



Melanomas may itch, bleed, ulcerate, or develop satellite lesions. Patients who present with metastatic disease or with primary sites other than the skin have signs and symptoms related to the affected organ systems. Melanoma is distinct from nonmelanoma skin cancers because it spreads locally, regionally, and distantly. An individual's risk of metastasis is directly related to the depth of invasion and ulceration of the primary lesion. Examining all lymph node groups is an important part of the physical examination.



Risk of melanoma:

- Positive family history: There is a 2.2-fold higher risk of developing melanoma with at least 1 affected family member.
- Pigmentary phototypes I or II: Including history of sunburns and history of high number of benign or dysplastic melanocytic nevi
- Immunocompromised: Such as post transplantation patients, patients with hematologic malignancy
- Excessive ultraviolet radiation exposure: Risks of melanoma increase with decreasing or increasing latitude.
- Social determinants of health: Lower socioeconomic status may be linked to more advanced disease at the time of detection. Several studies of newly diagnosed patients found that patients of low socioeconomic status have decreased melanoma risk perception, knowledge of the disease, and preventative practices.

<u>Evaluation</u>

Once a suspicious skin lesion is identified, refer to dermatology because a biopsy must be performed to confirm the diagnosis of melanoma. Obtaining a complete or excisional biopsy is preferred since the tumor staging of melanoma is based on the depth of invasion.

The following laboratory studies are indicated:

- Complete blood count
- Complete chemistry panel including alkaline phosphatase, hepatic transaminases, total protein, and albumin
- Lactate dehydrogenase

The following imaging modalities may be considered:

- Chest radiography
- Magnetic resonance imaging (MRI) of the brain
- Ultrasonography, possibly the best imaging study for diagnosing lymph node involvement

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

- computed tomography (CT) of the chest, abdomen, or pelvis
- Positron emission tomography (PET), PET-CT may be the best imaging study for identifying any sites of metastasis

Management

Melanoma is staged by the tumor, node, and metastasis (TNM) staging system guidelines. Treatment options and prognosis is based on stage.

Poor prognostic factors that affect survival in melanoma include:

- Tumor thickness, with a worse prognosis in thicker lesions
- Evidence of tumor in regional lymph nodes (i.e., stage III disease)
- A higher number of positive lymph nodes
- Presence of distant metastasis (i.e., stage IV disease)
- Anatomic site, with trunk or face lesions having a worse prognosis than extremity lesions
- Presence of ulceration
- Male sex

Management of malignant melanoma involves a combination of surgery, radiation therapy in very few cases, adjuvant immunotherapy, or targeted therapies. These interventions, which are proven to improve survival and long-term outcomes quality of life of a patient, along with the inability to complete the treatment course, which could result in an increased risk of recurrence and poorer survival outcomes. Therefore, awareness of potential complications of treatment is essential.

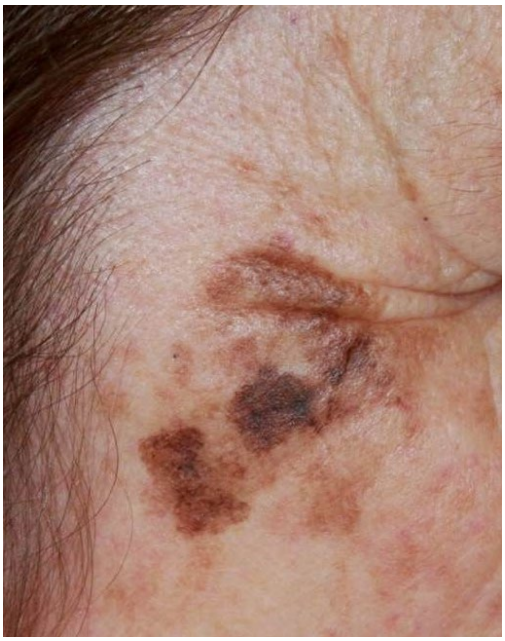
Comanage with dermatology and oncology as well as radiation oncology and surgery as clinically indicated. Since patients with a history of malignant melanoma have an approximately 9-fold increased risk of developing subsequent melanoma compared with the general population, refer to dermatology for a total body skin examination at least annually or more frequently as comanaged by dermatology.[23]

LENTIGO MALIGNA/LENTIGO MALIGNA MELANOMA

Characteristics

Lentigo maligna (LM) is a type of melanoma in situ that typically occurs in sun-damaged skin of the face and neck of older individuals between 65-80 years old. The surrounding skin typically shows evidence of chronic solar damage (solar elastosis, solar lentigines, or actinic keratoses). LM evolves slowly over many years and may progress to invasive lentigo maligna melanoma (LMM).

The time to progression of LM to invasive LMM ranges from less than 10 to more than 50 years with a lifetime risk of progression of ~5%.

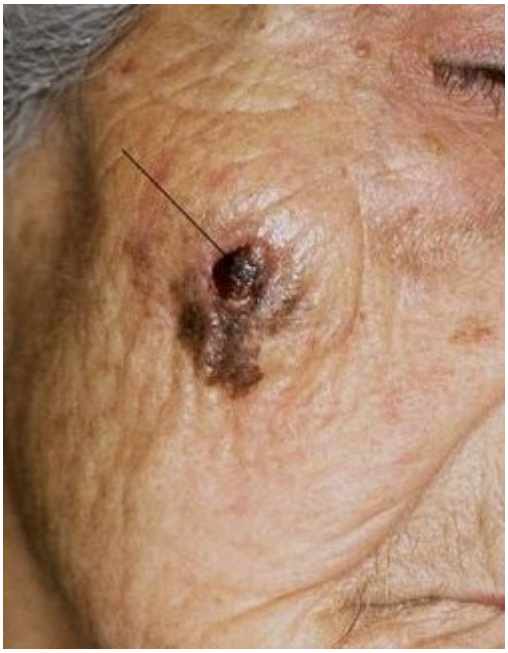


LM usually presents as an atypical, pigmented, macular lesion localized on severely sun-damaged skin of the head or neck. Infrequently, LM occurs in non-head and neck locations, such as the upper back, forearms, dorsum of the hands, and legs. LM are characterized by:

- Irregular shape
- Variable color, from light brown or tan to dark brown, black, pink, red, or white
- Variable size, from less than one to several centimeters
- Nonscaly smooth (nonpalpable) surface

Infrequently, LM occurs in non-head and neck locations, such as the upper back, forearms, dorsum of the hands, and legs.

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS



Consider LMM transformation with the development of:

- Darker asymmetric foci of pigmentation
- Color variegation
- Sharper borders
- Elevated or nodular areas
- Palpable subcutaneous firmness
- Ulceration or bleeding

LMM accounts for 10-15% of all melanomas.

Management

Refer patients with suspected LM/LMM to dermatology for surgical excision. Alternative nonsurgical treatments for LM include radiation therapy and topical immunomodulator therapy with imiquimod, which should be guided by dermatology. Nonsurgical therapies may be appropriate in older, frail patients with large lesions of the face that cannot be resected with adequate margins, when problematic reconstruction is anticipated, and in those who decline surgery. None of these treatment modalities has been compared with surgery in randomized trials.

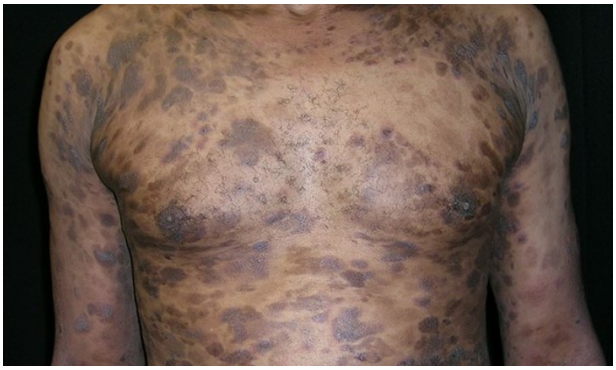
Comanage with dermatology and oncology as well as radiation oncology and surgery as clinically indicated. Since patients with a history of malignant melanoma have an approximately 9-fold increased risk of developing subsequent melanoma compared with the general population, refer to dermatology for a total body skin examination at least annually or more frequently as comanaged by dermatology.[23]

DERMATOLOGIC CONDITIONS: NEOPLASMS AND HYPERPLASIAS

CUTANEOUS T CELL LYMPHOMA

Characteristics

Cutaneous lymphoma is a type of non-Hodgkin lymphoma (NHL), most of which are T cell lymphomas or cutaneous T cell lymphomas (CTCL).

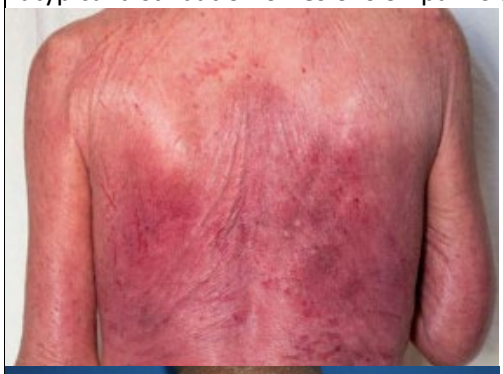


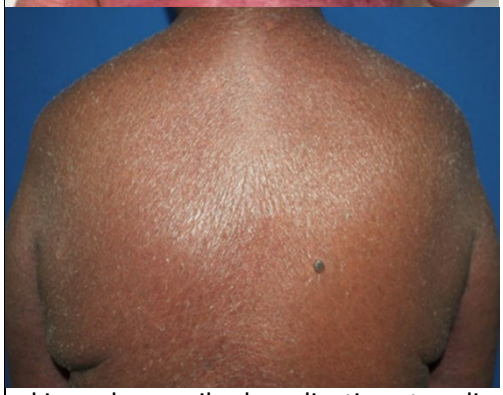
Most CTCL are mycosis fungoides (MF). Men are twice as likely as women to develop MF. The first sign of this disease is one or more patchy, scaly, red lesions on the skin. MF lesions can be very itchy. Often these lesions are the only symptom of MF. In more advanced disease, MF can progress to the plaque then tumor phase. Because MF can be confused with other skin problems, it can be hard to diagnose at first requiring biopsies. Advanced stages of MF cause diffuse skin involvement with ulcerations and metastasis.

In patients with lighter skin, MF typically presents as erythematous patches, plaques, or tumors seen in skin areas not typically exposed to sun. In patients with skin of color, there are different presentations of MF including hyperpigmented and hypopigmented lesions, lesions that present similarly to common dermatosis like psoriasis or lichen planus, or pruritus and secondary hyperpigmentation and lichenification. Morphology and distribution also differ on skin of color with a classic morphology which includes large lesions of polymorphic pigmentation, with discrete lesions of hypopigmentation and hyperpigmentation in areas such as the buttocks, breasts, and intertriginous regions. Morphology variations include a distribution of multiple hypopigmented patches on the trunk or extremities, or even a more atypical distribution of lesions on palms and soles.

HEALTH EQUITY ALERT

Patients with skin of color are impacted disproportionately by MF due to delayed diagnoses and limited research. The prevalence of MF varies, but studies suggest it is diagnosed in White patients at an older age (> 60 yo) and in Black patients at a younger age (> 40 yo) at advanced stages and with a more aggressive clinical course. Less responsive to treatments at advanced stages, many patients with darker complexions have worse survival rates than lighter-skinned patients. Therefore, knowing the dermatologic distinctions may improve MF recognition on skin of color.





Another type of CTCL is Sezary syndrome (SS), in which most or all of the skin is affected, instead of patches of skin. People with SS typically have a very itchy, scaly rash that can look like a severe sunburn on thickened skin (erythroderma). Lymphoma cells, called Sezary cells, can be found in the blood and lymph nodes. Whereas MF is usually slow growing, SS is an aggressive leukemic variant of CTCL. Other common cutaneous features of SS include alopecia, onychodystrophy, palmoplantar keratoderma, and ectropion. People with SS also often have further weakened immune systems, which increases their risk of serious infections.

Other types of CTCL are much rarer and not addressed in this care guide.

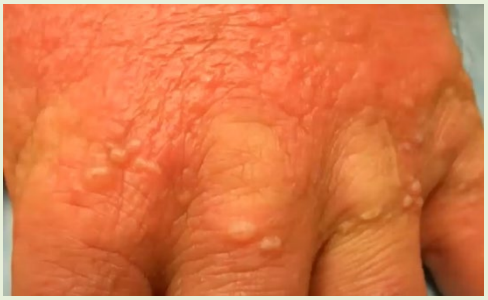
Management

Given the cutaneous and sometimes extracutaneous involvement characteristic of CTCL, these patients are often best managed in a multidisciplinary setting by dermatologists and oncologists in consultation with radiation oncologists.

The treatment options are based on the stage of the disease. Given the leukemic involvement in Sezary syndrome, the treatment is generally systemic, given alone or in combination with skin-based therapy. Patients usually have severe pruritus, which can be disabling. Thus, patients should be educated on moisturizing the skin and prescribed medications to relieve the itch. During treatment of the malignancy, quality of life should be a consideration since the outlook for most patients is guarded.[24]

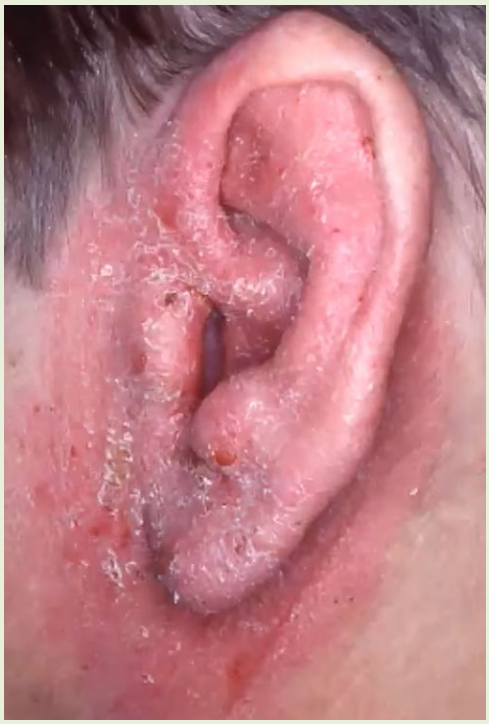
DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

ECZEMA



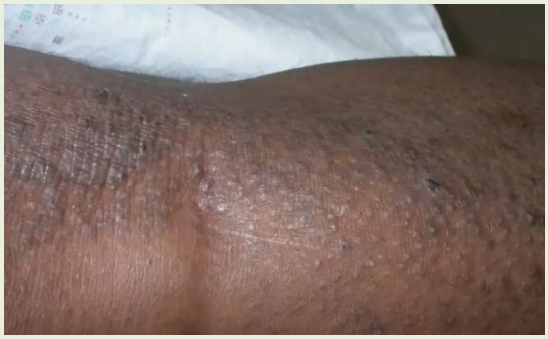
Eczema is an umbrella term, not a specific diagnosis, that refers to a group of conditions that cause inflamed skin with eczematous changes. Signs of inflamed skin include a popular or papulovesicular rash, pruritis, and excessive dryness. There are several types of eczema. *Atopic dermatitis* is the most common diagnosis. The other diagnoses of different types of eczema are *irritant contact dermatitis*, *allergic contact dermatitis*, *nummular eczema*, *dyshidrotic eczema*, *lichen simplex chronicus*, *seborrheic dermatitis*, *xerotic dermatitis*, and *stasis dermatitis*. Biopsy is limited in distinguishing among the different types of eczema.

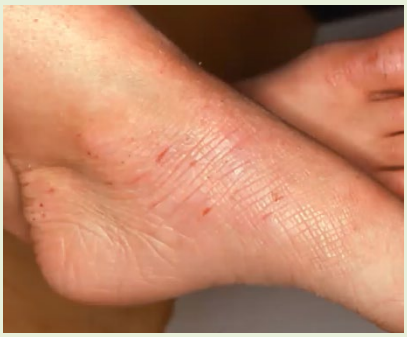
Acute eczema, which can occur with this example of irritant contact dermatitis, for example, presents with microvesicles and erythematous papules that coalesce into a plaque.



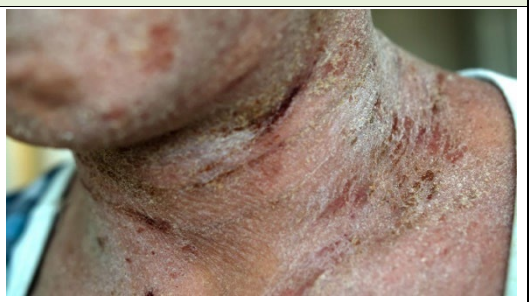
Subacute eczema, as display in this example of allergic contact dermatitis, may show more epidermal disruption with weeping, scaling, crusting, and erosions from scratching that has ruptured the microvesicles. Scratching also leads to excoriations.

Chronic eczema presents with lichenification from long-standing scratching and rubbing. Lichenification can look like accentuated skin markings and/or flat-topped papules.





ATOPIC DERMATITIS



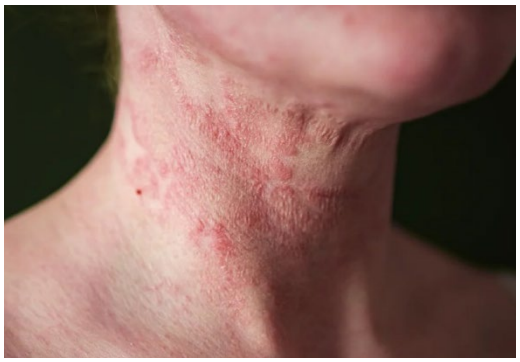
Characteristics

Atopic dermatitis (AD), which is a specific form of eczema, is the most common chronic inflammatory skin disease. AD has a complex etiology including genetic and environmental factors which lead to abnormalities in the epidermis and the immune system. AD is a type I hypersensitivity reactions mediated by IgE antibodies.

AD is seen in approximately 10% to 30% of children and 2% to 10% of adults in developed countries. This prevalence has increased two to three-fold in recent decades. AD has a higher incidence at higher latitudes, which may be related to decreased sun exposure and lower humidity levels. AD is divided into three subsets based on the age of onset:

- Early-onset AD (birth to 2 years old): most common type of AD, with approximately 60% of cases starting by age 1. Sixty percent of cases resolve by 12 years old
- Late-onset AD: symptoms begin after the onset of puberty
- Senile onset AD: an unusual subset with onset in patients older than 60 years old.

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS



A provider should take a patient's history and focus on the following:

- Onset and distribution of lesions with a relapsing and remitting course
- Severity of pruritus (e.g., keeping patient awake at night)
- Family and/or personal history of the atopic disease (AD, asthma, allergic rhinosinusitis, food allergy)
- Presence of contact allergens
- The presence of triggers including allergens (e.g., dust mites, animal dander), hot showers/sweating, soaps, fragrances, food hypersensitivities, and synthetic fabrics such as polyester.

Classic physical exam findings depend on age group but patients will present with itchy papular or papulovesicular rash over flexural surfaces. Adults have chronic lichenified (enhanced skin markings) lesions that have a predilection for hands. Individual lesions may also be further classified into acute (edematous, erythematous papules and plaques and/or vesicles/crusting), subacute (erythema, scale, variable crusting), or chronic (thick plaques with lichenification and scale) stages. The presence of associated findings (e.g., keratosis pilaris) may facilitate diagnosis.

Management

A biopsy is rarely needed, and a diagnosis can be made based on history and physical exam findings. The four major components of treatment include:

- Trigger avoidance
- Daily skin care
- Anti-inflammatory therapy
- Other complementary modalities

For the measurement of disease severity, several scales were identified without a single gold standard. Therefore, the American Academy of Dermatology (AAD) does not recommend the use of disease severity measurement scales or patient quality of life measurement scales for the general management of patients with AD. Providers should ask general questions about itch, total body surface area affected, sleep, impact on daily activity, and persistence of disease.

Daily skin care includes the application of emollients twice daily, with the application within three minutes of exiting lukewarm shower or bath to prevent skin drying. Ointments are the most occlusive but may be greasier. Topical corticosteroids, which should be applied before emollients to "lock-in" their effect, are first-line agents for acute flares. The potency should be strong enough to control a flare quickly, and consideration should be given for tapering every other day and for maintenance therapy twice weekly (e.g., weekends) using a medium potency topical corticosteroid in the usual areas of involvement to reduce disease flares and relapse. Reversible side effects of steroid use include skin atrophy and telangiectasia.

Sensitive areas (including the intertriginous areas of the axilla and groin, in addition to the face) may require topical nonsteroidal agents including calcineurin inhibitors such as tacrolimus and pimecrolimus. Newer non-steroidal agents include crisaborole, which is a topical phosphodiesterase-4 (PDE4) inhibitor ointment to tread mild-to-moderate AD. Topical roflumilast cream, another PDE4 inhibitor, is FDA approved for mild-to-moderate AD. Topical ruxolitinib, a Janus kinase (JAK) inhibitor, was also approved by the US Food and Drug Administration (FDA) for the treatment of mild-to-moderate AD but only for short-term, non-continuous treatment for patients with mild-to-moderate AD.

When AD is not controlled with topical agents, consider referring to dermatology and/or allergy and immunology, who comanage use of systemic agents including biologic therapy, JAK inhibitors, cyclosporine, phototherapy with narrowband UVB, azathioprine, mycophenolate mofetil, and methotrexate. Dupilumab is an injectable biologic therapy administered subcutaneously to treat moderate-to-severe, resistant, AD unresponsive to conventional therapy, including topical corticosteroids. Newer biologics include tralokinumab and lebrikizumab. JAK inhibitors, such as abrocitinib, upadacitinib, and baricitinib, are oral therapies to treat moderate-to-severe, resistant, AD unresponsive to conventional therapy with

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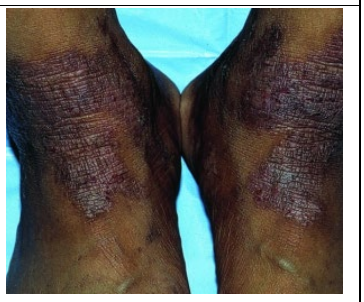
biologic therapy. Before starting JAK inhibitors in lieu of biologic therapy, careful patient selection is required given the cost of more potential adverse events. Lastly, systemic cyclosporine is indicated for short-term treatment for the most severe AD unresponsive to conventional therapy with biologic therapy. Like phototherapy, azathioprine, methotrexate, and mycophenolate, cyclosporine is only conditionally recommended for use.

Certain therapies are not recommended, such as systemic corticosteroids. Additionally, regular use of topical antimicrobials and topical antihistamines is not recommended.

Mild AD	Moderate AD	Severe AD
Over-the-counter moisturizer (fragrance-free is preferred if available) from the canteen		
Topical mid-/high-potency corticosteroid daily	Topical mid-/high-potency corticosteroid daily	Topical mid-/high-potency corticosteroid daily
	Topical low-/mid-potency corticosteroid with occlusive dressing	Topical low-/mid-potency corticosteroid with occlusive dressing
Add topical calcineurin inhibitor	Add topical calcineurin inhibitor	Add topical calcineurin inhibitor
Add crisaborole	Add crisaborole	
	Add dupilumab or tralokinumab	Add dupilumab or tralokinumab
	Replace dupilumab or tralokinumab with JAK inhibitor or cyclosporine	Replace dupilumab or tralokinumab with JAK inhibitor or cyclosporine

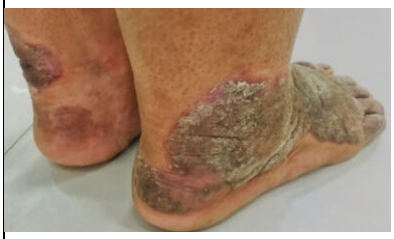
AD is associated with select allergic, atopic, immune-mediated, mental health, and bone health comorbidities and skin infections. There is also evidence associating AD with substance use, attention deficit hyperactivity disorder (ADHD), and metabolic syndrome.[25, 26, 27]

LICHEN SIMPLEX CHRONICUS (LSC)



Characteristics

Lichen simplex chronicus (LSC), also known as neurodermatitis, is a type of eczema that is considered a complication of other pruritic dermatologic conditions, as it often develops as a secondary condition in people with atopic dermatitis due to the chronic itching, leading to repetitive scratching that results in thickened, lichenified skin patches in localized areas.



LSC presents as dry, patchy areas of skin that are scaly and thick. The hypertrophic epidermis generally seen is typically the result of habitual scratching or rubbing of a specific area of the skin. The root of the disorder may be both a primary symptom, reflective of perhaps a psychological component (such as anxiety, depression, obsessive-compulsive disorder) or secondary to other cutaneous issues such as atopic dermatitis or psoriasis.

Management

Since LSC is a secondary skin disorder that results from excessive scratching, the treatment centers on the discontinuation of the itch-scratch cycle. Commonly used therapies include topical corticosteroids under occlusion and intralesional corticosteroids. Moisturizer and cold compresses can also control the itch.[25, 27]

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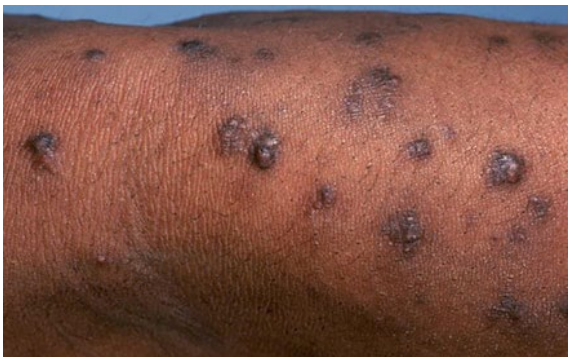
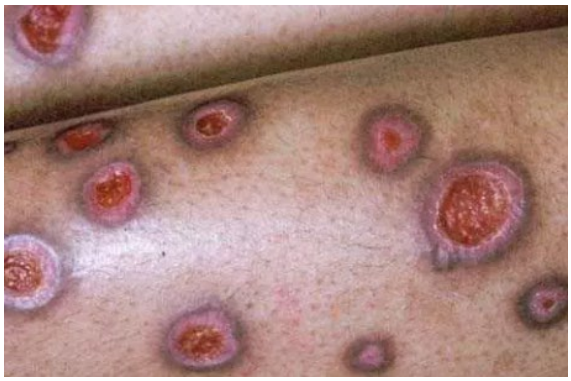
PRURIGO NODULARIS





Characteristics

Atopic dermatitis (AD) and prurigo nodularis (PN) are chronic dermatological conditions marked by severe itching and the presence of eczematous lesions such as papules and nodules. Both diseases can pose significant physical and psychological harm, leading to poor quality of life. In fact, AD can be an initial trigger for PN, and a patient can have both conditions at the same time. The close association between AD and PN suggests a multifaceted overlap and potential bi-directionality in developing these skin conditions.

PN is characterized by multiple, firm papules, plaques, and nodules predominantly found on the extensor surfaces of the extremities. These lesions, which can be flesh-colored, pink, or dusky purple, are intensely pruritic and manifest across diverse age groups.

Management

Management of PN encompasses a multidisciplinary approach with dermatology and mental health, particularly for patients with any underlying compulsive behaviors, such as skin picking. Long-term therapy is often necessary as PN tends to be refractory to conventional treatments, emphasizing the importance of patient education, counseling, and compliance.

Nemolizumab, a humanized monoclonal antibody against the receptor of IL-31, a pruritogenic cytokine, was approved by the US Food and Drug Administration (FDA) for the treatment of PN.

Gentle skin care using mild cleansers for bathing or showering and applying emollients multiple times per day to soothe the skin and reduce dryness is emphasized. To minimize the consequences of scratching and avoid excoriations, patients should keep their nails short and may wear gloves at night. Occluding the involved areas with dressings over cooling moisturizers or lotions may provide further relief.

Pharmacologic therapy with first-generation sedating antihistamines, such as hydroxyzine, administered at bedtime may be useful in controlling nocturnal pruritus. Both selective serotonin reuptake inhibitors and tricyclic antidepressants are also employed for chronic pruritus, especially when a component of depression is present.

For patients with a limited number of nodular lesions, start a super potent topical corticosteroid, such as clobetasol dipropionate 0.05% ointment, under occlusion once at nighttime for at least 2-4 weeks. Once pruritic is controlled, topical corticosteroids can be tapered to once or twice weekly and continued as a long-term maintenance regimen.

Consider referring to dermatology for widespread or recalcitrant disease, since systemic therapies, such as methotrexate, cyclosporine, or dupilumab, are required for such cases.[25, 27]

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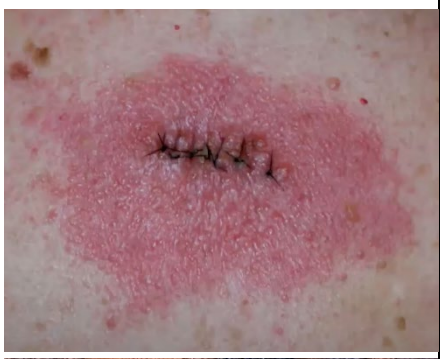
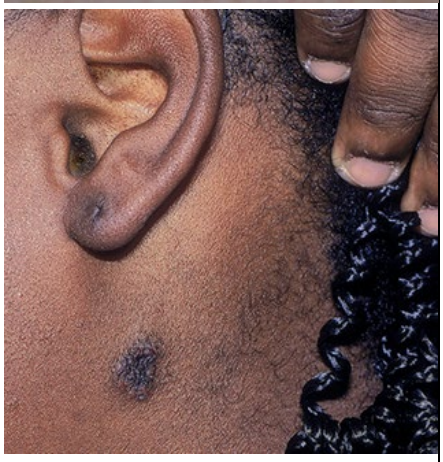
ALLERGIC CONTACT DERMATITIS





Characteristics

Allergic contact dermatitis (ACD) is the classic presentation of a T cell-mediated, delayed-type hypersensitivity response to exogenous agents and can occur even after years of uneventful use of a substance, such as neomycin or nickel. Acute dermatitis usually develops 48-72 hours after exposure. Patient who present with acute clinical manifestations of ACD present with skin lesion that are pruritic, erythematous, and indurated. For those with severe acute ACD, weeping vesiculation and bullae may occur. The reaction is generally limited to the site of contact and is associated with a sensation of burning, stinging, or pain. However, patchy or diffuse distributions may occur, depending upon the nature of the allergen or secondary transfer of the allergen from the primary site of contact to distant skin areas. Chronic ACD results from repeated exposure is characterized by skin that is dry, scaly, and hyperkeratotic. Excoriations, lichenification, and fissuring develop with long-standing ACD.

ACD (type IV hypersensitivity) and atopic dermatitis (AD) (type 1 hypersensitivity) are two different eczematous conditions that may coexist in the same patient. However, a 2017 meta-analysis of 74 studies did not find a difference in the frequency of ACD between individuals with AD and those without.

Common contact allergens include plant allergens, such as plants of the genus *Toxicodendron* genus (poison ivy, poison oak, poison sumac), which is also responsible for many cases of airborne ACD, due to exposure to pollen or other particles suspended in the air. Other common contact allergens are metals, fragrances, acrylates, medicaments, and preservatives. Nickel is found in alloys and plated objects, including jewelry, buttons, zippers, coins, keys, and some handcuffs.

Management

The morphology, regional distribution, and temporal course of dermatitis frequently suggest the diagnosis of ACD. The source of contact with allergens may be identified by reviewing the patient's activities, including occupation and hobbies. Products and objects for personal use, including prescription or over-the-counter topical preparations, cosmetics and toiletries, hair dyes, fragrances, eyeglasses, gloves, and clothing, should be reviewed. A history of long-term exposure to an allergen does not rule out ACD, since multiple exposures are typically necessary for sensitization and dermatitis to occur.

When the possible offending allergen is identified on the basis of clinical features and history, response to empiric therapy may avoid the need for patch testing. Improvement or resolution of the dermatitis with allergen avoidance and empiric treatment supports the diagnosis of ACD. Patch testing is an essential investigation in patients with persistent eczematous eruptions when contact allergy is suspected or cannot be ruled out. Patch testing helps to identify allergens that should be avoided. Skin prick testing (SPT) and radioallergosorbent testing (RAST) are types of allergy tests that checks for IgE-medicated type 1 hypersensitivity and, therefore, are **not** indicated to check for ACD.

Successful allergen avoidance can result in clearance of ACD. For occupational exposure, protective gloves can reduce or eliminate the exposure of the hands. Gloves are ideally selected based on the glove permeation and degradation properties, the nature of the compounds that are manipulated, and risk of ACD from materials used in the gloves. Rubber (latex) gloves contain allergens that may worsen hand dermatitis in some patients. Also, improperly chosen gloves may actually allow allergen penetration and trap the allergen against the skin. Education on the proper way to don and remove gloves is required to minimize exposure.

Advise patient to wash hands with lukewarm water and small amounts of mild, fragrance-free, and soap-free skin cleansers. Rinse and dry hands thoroughly and gently after washing. Use gentle emollients and moisturizers multiple

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

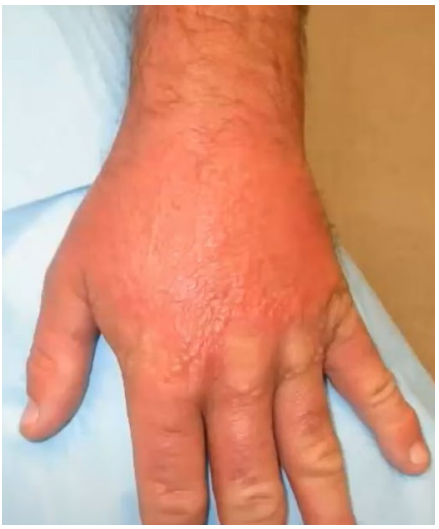
times per day and after hand hygiene. Thick petrolatum-based products are preferred to emollients containing lanolin or fragrances to reduce the risk of contact sensitization. Since irritant contact dermatitis (ICD) is often associated with or precedes the development of ACD, prework and after work emollients appear to confer some degree of protection against ICD. For acute, localized ACD, use topical corticosteroids and topical calcineurin inhibitors based on severity and location of dermatitis. For extensive, severe, or disabling ACD, systemic corticosteroids, such as prednisone 0.5 to 1 mg/kg per day (maximum 60 mg/day) for 7 days, is first-line treatment, especially if ACD involves >20% of the total body surface area or involving the face, hands, feet, or genitalia. The systemic corticosteroid dose may be reduced by 50% over the next 5-7 days and then tapered and discontinued over the following 2 weeks.

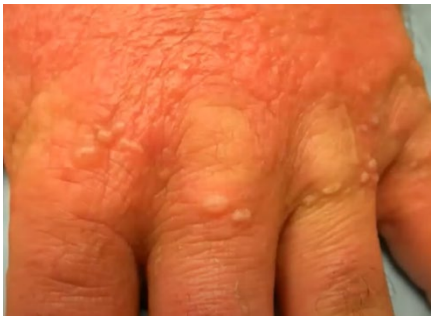
Skin atrophy is a complicating factor in many cases of chronic ACD and is exacerbated by overuse of topical corticosteroids so minimize continuous use of these agents beyond 2-4 weeks. Allergen-free emollients may be liberally used as an adjunct to active topical therapies in the treatment of ACD, particularly chronic, lichenified dermatitis.

Consider referring to allergy and immunology specialty if systemic immunosuppressive agents, such as methotrexate, azathioprine, mycophenolate mofetil, and cyclosporine, are needed for the management of certain cases of chronic ACD, such as airborne plant ACD from *Compositae* (e.g., parthenium, ragweed, aster, sunflower, chrysanthemum, artichoke) and *Toxicodendron* (poison ivy, poison oak, poison sumac) or photodermatitis, where allergen avoidance is impossible.

Persistence of dermatitis despite dutiful allergen avoidance suggests an alternative diagnosis, such as a systemic medication allergy or cutaneous lymphoma, or a comorbid diagnosis, such as AD.[28]

IRRITANT CONTACT DERMATITIS





Characteristics

Irritant contact dermatitis (ICD) is a localized, inflammatory eczematous skin response to a wide range of chemical or physical agents. Unlike atopic dermatitis (AD) or allergic contact dermatitis (ACD), ICD is not due to a hypersensitivity reaction and results from direct cytotoxic effect of irritants.

Everyone has a threshold of ICD, but patients with AD have a lower threshold of irritation. In fact, ICD, ACD, and AD may coexist in the same patient.

Water is a common irritant. Other common chemical irritants include detergents and surfactants, solvents, oxidizing agents, acids, and alkalis. Physical irritants include metal tools, wood, fiberglass, plant parts, paper, and dust or soil.

The clinical manifestations of ICD range from mild skin dryness and erythema to acute or chronic eczematous dermatitis and even skin necrosis from a chemical burn. Acute ICD results from a single exposure to an irritant or caustic chemical causing erythema, edema, vesicles, bullae, and oozing. The reaction is generally limited to the site of contact and is associated with a sensation of burning, stinging, or pain. Chronic ICD results from repeated exposures to mild irritants or low concentrations of strong irritants, and is characterized by erythema, scaling, lichenification, hyperkeratosis, and fissuring. Patients may complain of burning, pain, and itching.

Management

The diagnosis of ICD is based upon the clinical finding of a localized dermatitis in a patient with a history of exposure to chemical or physical irritants. For chronic ICD, skin patch testing may be performed to exclude ACD. As ACD and ICD may coexist, skin patch testing should also be considered when dermatitis fails to respond to appropriate treatment and when a significant allergen exposure exists. Although not routinely performed for the diagnosis of ICD, consider referring to dermatology for a skin biopsy if necessary to exclude other skin disorders.

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Treatment of ICD includes:

- Identification and avoidance of the offending irritant(s)
- Treatment of skin inflammation
- Restoration of the epidermal barrier function
- Prevention of further exposure

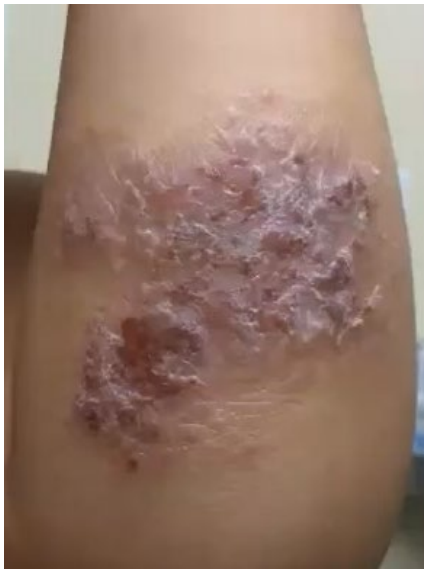
Advise patient to wash hands with lukewarm water and small amounts of mild, fragrance-free, and soap-free skin cleansers. Rinse and dry hands thoroughly and gently after washing.

Use gentle emollients and moisturizers multiple times per day and after hand hygiene. Thick petrolatum-based products are preferred to emollients containing lanolin or fragrances to reduce the risk of contact sensitization. Since ICD is often associated with or precedes the development of ACD, prework and after work emollients appear to confer some degree of protection against ICD. Use in combination with topical corticosteroids based on severity and location of dermatitis. Calcineurin inhibitors have not been proven effective in the treatment of ICD and, in some studies, have been found to be irritants.[29,30]

NUMMULAR ECZEMA

Characteristics

Nummular eczema, also called discoid eczema, is a chronic, inflammatory skin disease characterized by multiple pruritic, coin-shaped, eczematous lesions ranging in diameter from 1-10 cm usually involving the extremities. In the acute phase, lesions are dull red to purple, exudative, and crusted. Over time, the lesions become drier and scalier, occasionally with central clearing leading to annular lesions.

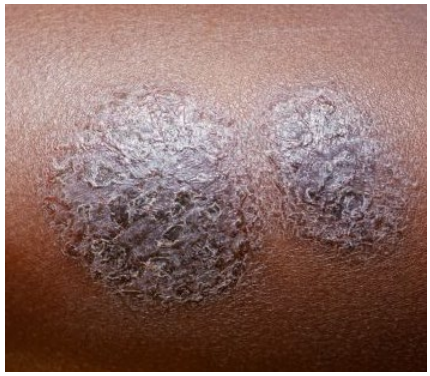


The legs and the upper extremities are the sites most frequently involved. Involvement of the trunk is less common, but when present, the lower trunk is more likely to be involved than the upper trunk. If the face or neck is involved, alternative diagnoses should be considered.

Nummular eczema is a chronic and relapsing disease. Days to months after resolution, apparently dormant lesions may become active again or new lesions may occur in adjacent areas. Nummular eczema can co-occur with atopic dermatitis (AD). It often affects older patients, men more often than women.

Management

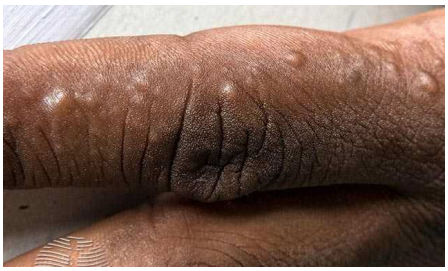
Daily skin care includes the application of emollients twice daily, with the application within three minutes of exiting lukewarm shower or bath to prevent skin drying. Ointments are the most occlusive but may be greasier. High- or superhigh potency topical corticosteroids, which should be applied before emollients to "lock-in" their effect, are first-line agents for acute flares. Apply 1-2 times daily for 2-4 weeks under occlusive dressings. For recalcitrant isolated lesions, consider intralesional triamcinolone. Reversible side effects of topical corticosteroid use include skin atrophy, dyspigmentation, and telangiectasia. For severe or refractory disease, refer patients to dermatology. Other therapies to consider are phototherapy, systemic corticosteroids, cyclosporine, methotrexate, or dupilumab.[30]

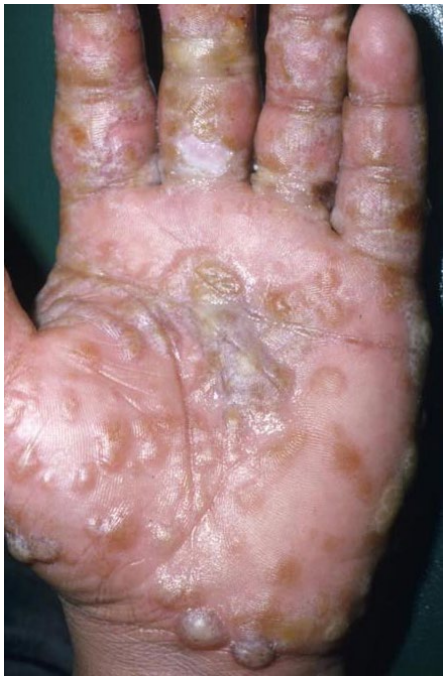


DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

DYSHIDROTIC ECZEMA

Characteristics





Dyshidrotic eczema, also known as dyshidrosis or pompholyx, is characterized by an intensely pruritic vesicular eruption comprised of clear, deep-seated vesicles without erythema erupting on the lateral aspects of fingers, the central palm, and/or plantar surfaces. Vesicles and bullae persist for several weeks, desiccate, and resolve with desquamation. Recurrence is common, and patients typically experience frequent episodes every 3-4 weeks for months or years. Between episodes, the skin may return to a normal appearance, in contrast to the persistent signs and symptoms of chronic vesicular hand dermatitis secondary to irritant contact dermatitis, allergic contact dermatitis, or atopic dermatitis. However, frequent relapses may result in chronic changes characterized by red, lichenified, and scaling patches or plaques with fissures.

Dyshidrotic eczema is severe if it involves the entire palmar or plantar surface and presents with large vesicles or bullae that are disabling. Disabling is defined as lesions that prevent walking or use of the hands or lesions that are intensely painful or pruritic regardless of size. Severe episodes can affect the nail matrix and produce dystrophic nail changes such as horizontal ridging and color changes.

Dyshidrotic eczema most commonly affects young adults, especially women.

Management

Consider referral to dermatology if the diagnosis is uncertain. Potential indications for skin biopsy include lack of response to treatment and exclusion of other conditions in the differential diagnosis. If a skin biopsy is performed, periodic acid-Schiff (PAS) staining for fungal elements may be useful in excluding a fungal infection, and direct immunofluorescence may be useful to confirm a diagnosis of bullous pemphigoid.

Also consider referral to dermatology if patch testing is warranted in the setting of patients who do not respond to initial therapy since there may be a component of allergic contact dermatitis.

Treatment of dyshidrotic eczema includes:

- Identification and avoidance of the offending factors
- Treatment of skin inflammation
- Restoration of the epidermal barrier function
- Prevention of further exposure

Advise patient to wash hands with lukewarm water and small amounts of mild, fragrance-free, and soap-free skin cleansers. Rinse and dry hands thoroughly and gently after washing.

Use gentle emollients and moisturizers multiple times per day and after hand hygiene. Thick petrolatum-based products are preferred to emollients containing lanolin or fragrances to reduce the risk of contact sensitization. Advise patients to wear protective gloves to avoid cold, friction, and irritant exposure. Use in combination with high- or superhigh potency topical corticosteroids based on severity. Topical corticosteroids are applied twice daily for two to four weeks. Ointments are generally preferred to other vehicles (creams, solutions, or foams) because they contain fewer potential irritants and allergens, such as additives or preservatives. Reversible side effects of topical corticosteroid use include skin atrophy, dyspigmentation, and telangiectasia.

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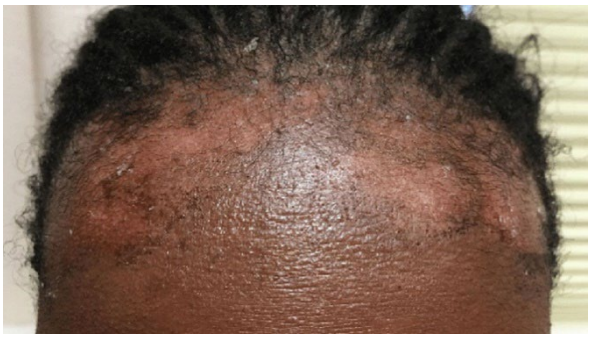
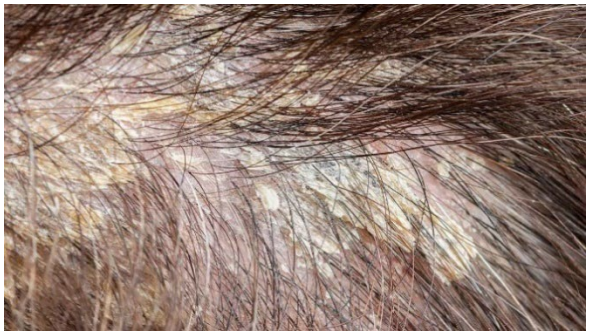
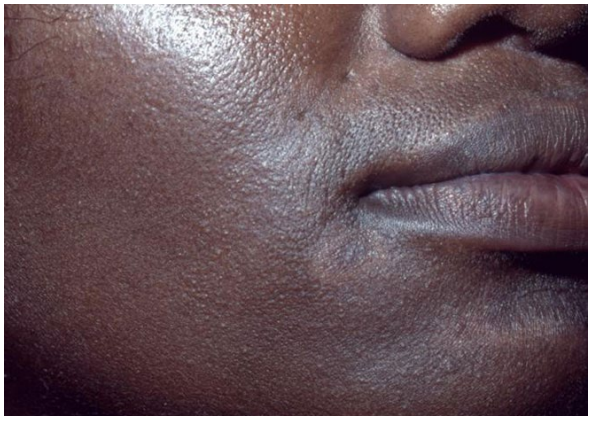
If long-term use of topical corticosteroids is avoided, calcineurin inhibitors is an alternative treatment using tacrolimus 0.1% ointment is applied twice daily until resolution.

For severe disease, consider a short course of systemic corticosteroids and refer to dermatology. Start with prednisone 40-60 mg daily for a week. If there is an adequate response, the dose is then reduced by half in the next 5-7 days. Continue to taper and discontinue over the following 2 weeks.[30]

SEBORRHEIC DERMATITIS







Characteristics

Seborrheic dermatitis is a common form of chronic, relapsing eczema that usually affects the scalp, though it can affect other parts of the body as well. Affected individuals are usually healthy, although seborrheic dermatitis has been associated with HIV, Parkinson disease and other neurologic disorders, and use of neuroleptic medications.

The mildest and most common form of seborrheic dermatitis is dandruff, also known as pityriasis sicca, in which the scalp shows fine, white, diffuse scaliness without underlying erythema. Dandruff may be asymptomatic or accompanied by mild pruritus.

More severe forms of scalp seborrheic dermatitis present with visible inflammation, consisting of patchy, orange to salmon-colored or grayish plaques covered with yellowish, greasy scales (pityriasis steatoides). Other affected areas include face, especially periocular, and trunk.

Management

For dandruff, use medicated shampoos containing zinc pyrithione, selenium sulfide, salicylic acid, coal tar, or ketoconazole in combination with low-potency topical corticosteroids. Topical antifungal agents are well established in the treatment of seborrheic dermatitis of the scalp and face because of their ability to decrease the population of *Malassezia furfur* on the affected skin and their anti-inflammatory property. Topical roflumilast 0.3% foam, approved in 2024 by the US Food and Drug Administration (FDA), treats seborrheic dermatitis.

For severe cases, refractory cases, or seborrheic dermatitis causing blepharitis, consider referral to specialty care. Also consider alternative diagnoses for severe or refractory cases, since these may be due to dermatophytosis, like tinea capitis, which is treated with systemic antifungal therapy and does not respond to topical therapy alone.[31]

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

ADNEXAL SKIN CONDITIONS

In dermatology "adnexal" refers to the appendages of the skin, which includes sweat glands (both eccrine and apocrine), sebaceous glands, and hair follicles. Each of these glands plays a specific role in skin function:

- **Eccrine glands:** Produce watery sweat that helps regulate body temperature.
- **Apocrine glands:** Located in areas like the armpits and groin, produce a thicker, protein-rich sweat that can contribute to body odor.
- **Sebaceous glands:** Part of the pilosebaceous unit, secrete sebum, an oily substance that lubricates the skin.
- **Hair follicles:** The hair bulb in the lower portion of the hair follicle and the hair shaft make up part of the pilosebaceous unit

This section will address diseases of the eccrine, apocrine, and sebaceous glands. Disorders of hair will be covered in a different section.

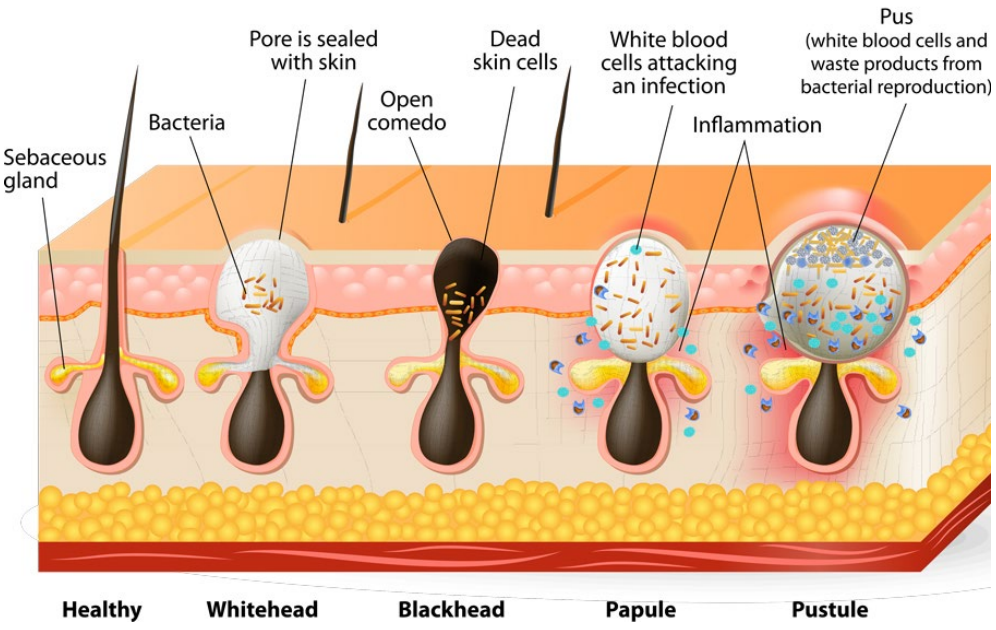
ACNE VULGARIS

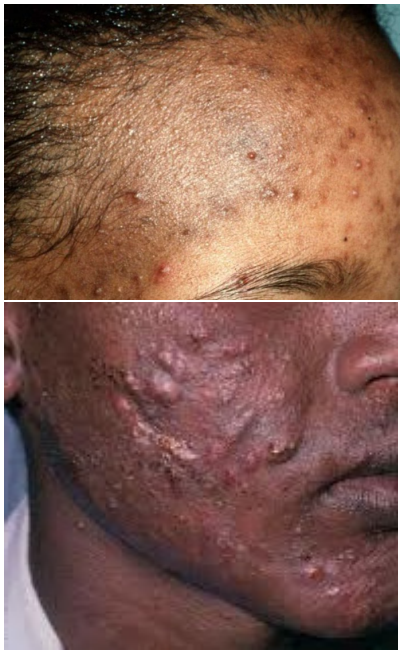
Characteristics

Acne vulgaris, or simply acne, is an inflammatory disorder of the pilosebaceous unit, which is comprised of the hair follicle and sebaceous gland. It is a common cutaneous disorder due to characterized by chronic or recurrent development of papules, pustules, or nodules on the face, neck, trunk, or proximal upper extremities, which correlates with areas of the body with large, hormonally responsive sebaceous glands.

One or more types of active lesions may be present, including:

- Closed comedones or “whitehead” – Noninflammatory; <5 mm; dome-shaped; smooth; skin-colored, whitish, or grayish papules.
- Open comedones or “blackhead” – Noninflammatory, <5 mm papules with a central, dilated, follicular orifice containing gray, brown, or black, keratotic material.
- Papulopustular acne – Inflamed, relatively superficial papules and pustules, typically <5 mm in diameter.
- Nodular acne – Deep-seated, inflamed, often tender, large papules (≥0.5 cm) or nodules (≥1 cm). Nodular acne is sometimes inaccurately referred to as "cystic" or "nodulocystic" acne, but true cysts are rare.





Skin pigmentation may mask the characteristic erythema of inflamed lesions in patients with highly pigmented skin. The extent and severity of skin involvement varies widely, ranging from the periodic appearance of a few small comedones to the chronic presence of numerous inflamed nodules involving the majority of skin in an affected region.

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

Associated factors and comorbidities for acne include:

- Repeated mechanical trauma that rupture comedones
- Increased milk consumption and high glycemic diets
- Periods of high stress
- Insulin resistance
- Hyperandrogenism from polycystic ovarian syndrome, late-onset adrenal hyperplasia, and adrenal or ovarian tumors

Resolution of individual acne lesions may leave transient or permanent changes on the skin, such as post inflammatory erythema, hyperpigmentation, and scarring. The risk for hyperpigmentation increases with increasing baseline skin pigmentation and is particularly common in individuals with pigmentary phototypes IV to VI. Inflammatory acne is considered more likely to result in scarring than noninflammatory acne. Various types of scars can result from acne vulgaris, including atrophic scars (rolling scars, boxcar scars, and ice pick scars), hypertrophic scars, and keloids.

Management

Routine microbiologic testing is not recommended, but if patients exhibit acne-like lesions suggestive of Gram-negative folliculitis, consider microbiologic testing. Similarly, routine endocrinologic evaluation is not recommended, but laboratory evaluation is recommended for patients who have acne and additional signs of androgen excess. Consider referring to endocrinology for guidance on additional testing for patients with acne and signs of androgen excess.

Therapy choice may be influenced by age of the patient, site of involvement, extent and severity of disease, and patient preference. Daily skin care includes washing with a gentle cleanser and avoidance of topical products that irritate skin.

For topical therapy, recommend multimodal therapy using a combination of benzoyl peroxide, topical retinoids, and topical antibiotics. Topical antibiotic monotherapy is not recommended. When using topical antibiotic therapy, concomitant use of benzoyl peroxide prevents the development of antibiotic resistance. Fixed dose combinations are recommended for ease of use and adherence to appropriate therapy.

Other topical therapies include salicylic acid (a comedolytic agent) and azelaic acid (a comedolytic, antibacterial, and anti-inflammatory agent). Azelaic acid may be used on patients with sensitive skin and patients with darker skin due to the lightening effects on dyspigmentation.

For patients with moderate to severe acne vulgaris, use topical therapy with systemic therapy using systemic antibiotics, hormonal agents, or isotretinoin. Systemic antibiotics have been used to treat moderate to severe acne. Oral doxycycline and minocycline are commonly used. Limit the use of systemic antibiotics when possible to reduce the development of antibiotic resistance and other antibiotic-associated complications. Outpatient antibiotic stewardship promotes appropriate antibiotic use in which patients receive the right dose of the right antibiotic at the right time for the right duration, typically no more than 3 months.

Among hormonal agents, there are four FDA-approved combined oral contraceptives for treatment of acne in patients with a uterus, including formulary norgestimate/ethinyl estradiol and formulary norethindrone acetate/ethinyl estradiol. Spironolactone is not FDA-approved for the treatment of acne, but studies have demonstrated similar treatment effectiveness to systemic antibiotics. Finally, intralesional corticosteroid therapy has been used as an adjuvant therapy for acne in patients with larger acne papules or nodules. Use intralesional corticosteroid injections judiciously for patients who are at risk of acne scarring and/or for rapid improvement in inflammation and pain. Be cautious of localized skin atrophy, systemic absorption of steroids, and adrenal suppression.

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

Isotretinoin is recommended for patients with severe acne and for patients who have failed standard therapy with other topical or systemic treatment. Patients with acne and with psychosocial burden or scarring have severe acne and are candidates for isotretinoin. For patients undergoing treatment with isotretinoin, monitor LFTs and lipid panel. For patients of childbearing potential, pregnancy prevention is mandatory.[32]

Management of Acne Vulgaris

Baseline Evaluation

Severity Assessment

- Acne objective severity assessment using the Physician Global Assessment (PGA) at follow up
- Assess satisfaction with appearance, extent of scar, treatment satisfaction, long-term acne control, and impact on quality of life

Routine microbiological and endocrine testing not indicated

Mild

Topical Treatments
Recommend multimodal therapy combining multiple mechanisms of action

Multimodal Topical Therapy

- Topical retinoids
- Benzoyl peroxide
- Fixed dosages
  - Benzoyl peroxide + topical retinoid
  - Benzoyl peroxide + topical antibiotics
  - Topical retinoid + topical antibiotics
    Concomitant use of benzoyl peroxide can prevent development of antibiotic resistance
- Salicylic acid
- Azelaic acid

Moderate to Severe

Systemic Antibiotics
Limit systemic antibiotic use (<3 months), when possible, to reduce development of antibiotic resistance or other antibiotic-associated complications

- Doxycycline (*not as monotherapy*)
- Minocycline (*not as monotherapy*)
- *+ benzoyl peroxide and other topical therapy*

Hormonal Agents

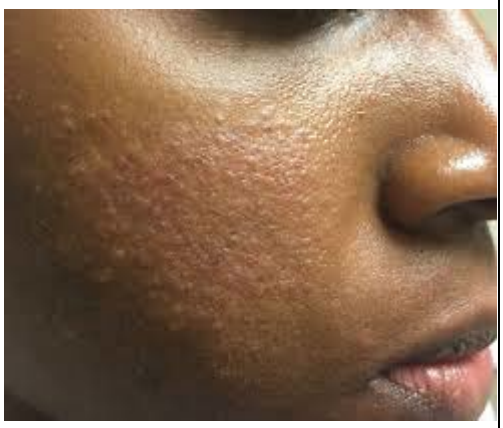
- Spironolactone
  Consider potassium monitoring for patients with risk factors for hyperkalemia (e.g., older age, comorbidities, other medications)
- Intralesional corticosteroids
  Adjuvant treatment for larger acne papules or nodules at risk of acne scarring or for rapid involvement in inflammation and pain

Isotretinoin
Consider daily isotretinoin for patients with psychosocial burden and/or scarring
Requires monitoring of LFTs and lipid panel
Pregnancy prevention mandatory

- Standard isotretinoin

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

ACNE ROSACEA



Characteristics

Acne rosacea is a common facial disorder presenting most commonly in adulthood, estimated to affect ~14 million Americans. This chronic disorder is characterized by intermittent periods of exacerbation. Clinical signs of rosacea include central facial erythema or duskiness, inflammatory lesions (papules, pustules), and telangiectasias. The most common clinical presentations of cutaneous rosacea include the inflammatory (papulopustular) and erythematotelangiectatic subtypes. Rough, dry, or scaly facial skin is a common feature. Other presentations include phymatous rosacea (such as rhinophyma) and granulomatous rosacea. Ocular rosacea is not uncommon in patients with cutaneous rosacea, and clinical presentations include conjunctivitis, blepharitis, stye formation, and keratitis.

Accurate diagnosis and prompt treatment prevent permanent scarring, persistent erythema, and ocular sequelae. Based on the latest diagnostic guidelines by the National Rosacea Society Expert Committee, one of the following clinical presentations is considered diagnostic for rosacea:

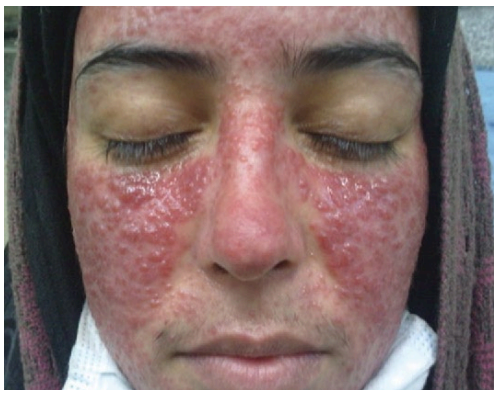
- Fixed centrofacial erythema in a characteristic pattern that may periodically intensify
- Phymatous changes

Two of the following major criteria below are also considered diagnostic:

- Flushing
- Papules and pustules
- Telangiectasia
- Ocular manifestations including lid margin telangiectasia, interpalpebral conjunctival injection, spade-shaped infiltrate in the cornea, and scleritis and sclerokeratitis; ophthalmic evaluation is necessary for patients with ocular symptoms

Management

Nonpharmacologic interventions for the management of the cutaneous manifestations of rosacea include avoidance of triggers of flushing, gentle skin care, sun-protection, and the use of cosmetic products. Triggers for flushing include:



- Extremes of temperature
- Sunlight
- Spicy foods
- Alcohol
- Exercise
- Acute psychologic stressors
- Certain medications
- Menopausal hot flashes

Practical measures, such as applying cool compresses and transferring to cool environments, help reduce flushing after encounters with triggers. In addition to midday sunlight avoidance and use of broad-spectrum sunscreen SPF 30 or higher, daily skin care includes washing daily with a gentle cleanser, application of emollients twice daily, and avoidance of topical products that may irritate the skin, such as toners, astringents, and chemical exfoliating agents.

Among topical therapy, avoid topical corticosteroids, which can exacerbate rosacea though rebound flaring or induction of rosacea-like perioral dermatitis. Treatment options are directed by the predominant type of rosacea to reduce inflammation.

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

Topical Treatment

Erythema

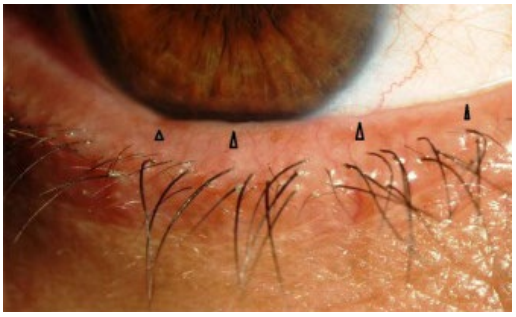
- Brimonidine tartrate (alpha-2 agonist) 0.33% gel (Daily application on the face)

Inflammatory papules and pustules

- Ivermectin 1% cream (daily application)
- Azelaic acid 15% gel (daily 1 to 2 times application)
- Metronidazole 0.75% gel, cream, or lotion and 1% gel or cream (daily 1 to 2 times application)
- Sulfacetamide 10% with sulfur 5% cream, lotion, emollient, suspension (daily 1 to 3 times application)
- Topical benzoyl peroxide 5%-clindamycin 1% cream (daily application) for second-line therapy
- Topical permethrin 5% cream for refractory rosacea diagnosed as demodicidosis (*Demodex folliculitis*)

Ocular Involvement

- Artificial tears
- Metronidazole 0.75% gel (daily 1 to 2 times application on eyelids) limited data available for efficacy
- Cyclosporine 0.05% eyedrops, (one drop every 12 hours) limited data available for efficacy



Systemic Treatment: used for flares that do not respond to topical treatment and moderate to severe disease

Flushing

- Propranolol (20 to 40 mg 2 to 3 times/day), carvedilol (6.25mg 2 to 3 times/day)

Inflammatory papules and pustules

- FDA approved subantibiotic dose oral doxycycline (SDD_{40}) [modified-release 40 mg daily (which is composed of 30 mg immediate-release and 10 mg delayed-release beads) for 8 to 12 weeks]
- Minocycline (50 to 100 mg twice daily for 8 to 12 weeks) + topical therapy
- Doxycycline (50 to 100 mg twice daily for 4 to 12 weeks) + topical therapy
- Tetracycline (250 to 500 mg twice daily for 8 to 12 weeks) + topical therapy
- Azithromycin (250-500 mg 3 times weekly for 4 to 8 weeks) + topical therapy
- Isotretinoin (0.25 to 0.3 mg/kg/day for 12 to 16 weeks)

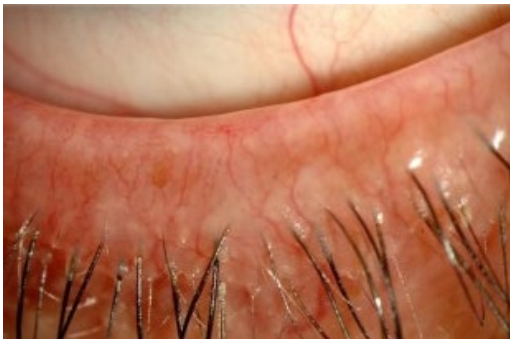
Phyma (inflamed)

- Doxycycline (100 mg 1 to 2 times daily for 8 to 12 weeks) + topical therapy
- Tetracycline (250 to 500 mg twice daily for 8 to 12 weeks) + topical therapy
- Isotretinoin (0.25-0.3 mg/kg/day for 3 to 4 months) if refractory to systemic antibiotics + topical therapy

Ocular Involvement

- SDD_{40} (40 mg daily for 8 to 12 weeks)

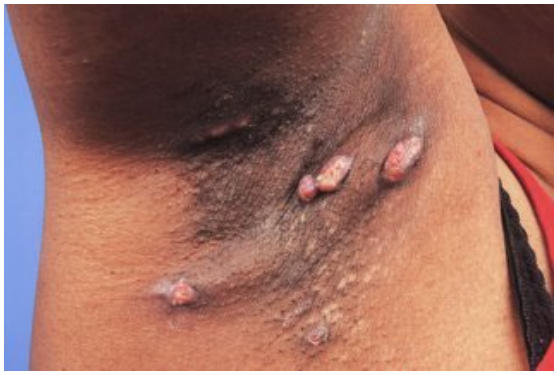
Procedural treatment includes intense pulsed light therapy and laser for erythema and telangiectasias, as well as advanced phymatous skin changes. Electrosurgery and surgical resection to debulk advanced phymatous skin changes. However, these procedures have increased risk of pigmentary changes among patients with darker skin tones. Refer patients to dermatology for patients that do not resolve with systemic antibiotics + topical therapy and for patients with phymatous skin changes. Refer patients to ophthalmology for patients with ocular involvement.



Because rosacea is a chronic disorder and treatments are not curative, continued therapy is typically necessary to maintain treatment response. Most commonly, long-term topical therapy is administered. Subantimicrobial doses of doxycycline are also an option.[33]

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

HIDRADENITIS SUPPURATIVA



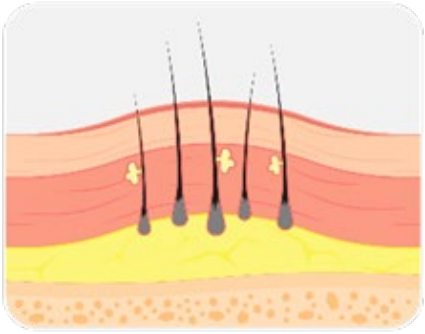
Characteristics

Hidradenitis suppurativa (HS), also called acne inversa, is a chronic, debilitating, inflammatory disease of the hair follicle. Patients present with painful inflammatory nodules, abscesses, comedones, scarring, and tunneling sinus tracts, with predilection for intertriginous areas of the body (most commonly the axillae and inguinal and anogenital regions).

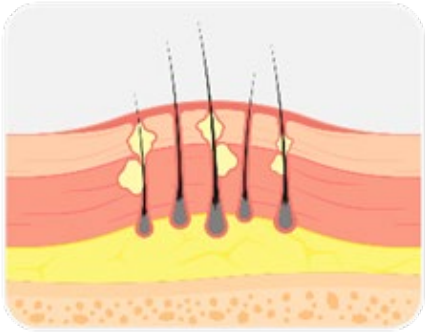
Diagnosis relies on clinical findings of

- typical HS lesions
- predilection for intertriginous sites
- recurrence

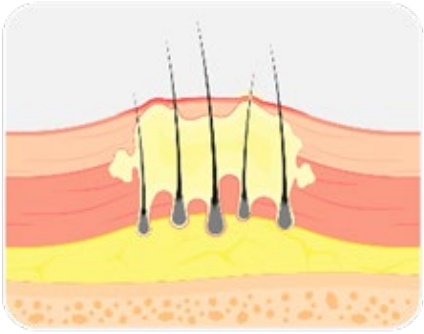
The prevalence of HS ranges from 0.1-2%, with predilection for patients who are in the third and fourth decades of life, female, and of lower socioeconomic status. HS also disproportionately affects Black patients. Clinical performance, Hurley staging, and inflammatory lesion counts (abscesses and inflammatory lesions) are used for grading and classification of HS. Consider clinically following pain using a validate pain scale.



STAGE I
Single or a few abscess formation without sinus tracts and scarring



STAGE II
Recurrent abscesses widely separated lesions with initial formation of sinus tract



STAGE III
Multiple interconnected sinus tracts and abscesses across entire are with evidence of scarring and purulence

Management

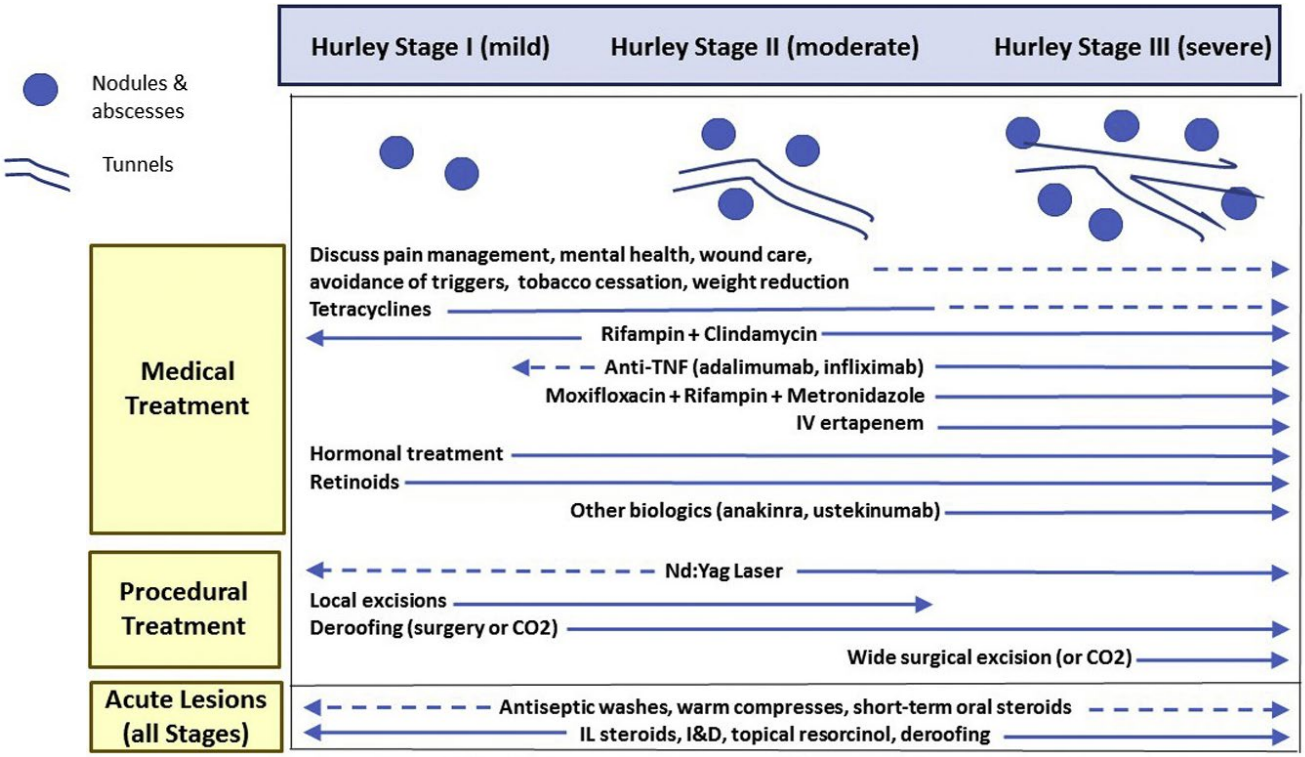
Microbiologic screening has limited utility in HS since mixed normal flora and skin commensals are the main bacteria cultured from suppurative discharge. Culture is not recommended unless signs of secondary infection, such as surrounding cellulitis or fever, are present.

HS comorbidities for which to screen routinely include smoking, diabetes, obesity, metabolic syndrome, depression or anxiety, follicular occlusion tetrad, and cutaneous squamous cell carcinoma of HS-affected skin. Recommend thorough review of systems, smoking history, Hgb A1c, weight, and periodic skin examination, particularly of chronic lesions on the perineum and buttocks, where squamous cell carcinoma is most common.

Recommend referral to dermatology for comanagement. For acute flares, use antiseptic washes, such as chlorhexidine, benzoyl peroxide, and zinc pyrithione. Topical and intralesional therapies are available for select lesions. However, systemic antibiotic monotherapy, usually tetracyclines for 12-weeks during acute flare or as long-term management as appropriate, is possible for mild disease. In advanced disease, systemic antibiotics are adjunctive due to recurrence.

Consider hormonal agents, such as combined oral contraceptives, spironolactone, metformin, and finasteride, in appropriate female patients, either as monotherapy for mild-to-moderate HS or in combination with other agents for more severe disease. Avoid progestogen-only contraceptives, which potentially worsen HS.

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS



Lastly, consider short-term pulse corticosteroid therapy for acute flares or to bridge patients to other treatment. Consider long-term systemic corticosteroids tapered to the lowest possible dose in cases of severe HS, as an adjunct therapy in patients with suboptimal response to standard therapy. Biologics, such as adalimumab, infliximab, anakinra, and ustekiunumab, are the cornerstone of therapy for moderate-to-severe HS.

For patients with recurrent nodules and tunnels, refer to surgery for deroofing of acute and chronic lesions. In contrast to deroofing, incision and drainage (I&D) has been associated with recurrence rates approaching 100%, although it provides acute relief when other methods are not feasible. Deroofing is a technique, in which abscesses and associated sinuses are probed and the skin overlying the sinus or abscess cavity is removed stepwise with the base left untreated. Deroofing small lesions with a punch tool or by other methods is preferred to simple drainage. For large nonrecurrent abscesses, I&D provides acute relief until deroofing can be done. Carefully prescribed short-acting opioid analgesics may be needed for severe pain during acute flare of HS. Consult Wound Central Team for appropriate management of nonsurgical and postsurgical HS wounds. For large open wounds, consider use of negative-pressure therapy for a short period (1-4 weeks), followed by delayed reconstruction with surgery.[34]

AUTOIMMUNE DISORDERS

Autoimmune disorders drive the inflammatory cascade which can affect the skin directly. This section will review the more common autoimmune diseases, distinctive characteristics, and appropriate therapies.

VITILIGO

Characteristics

Vitiligo is characterized by a focal or generalized distribution of depigmented macules and patches. The hairs in the vitiliginous areas are usually white. Vitiligo commonly occurs in periorificial areas (mouth, orbits, vagina, anus) or at sites of trauma (hands, elbows, knees).

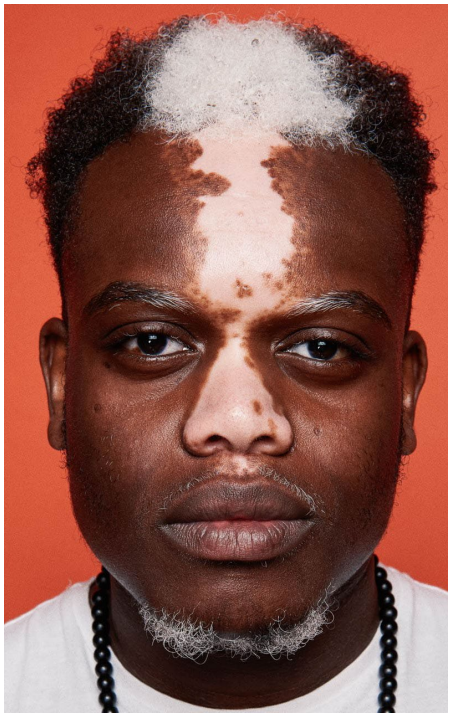
Note the sparing of the perifollicular skin of the glabella, which is common with vitiligo. Vitiligo exhibits koebnerization, with skin lesion often first affecting areas of frequent trauma, such as the fingertips and bony prominences.

Vitiligo has been linked to the same autoimmunity predisposition genes as those for pernicious anemia, autoimmune thyroiditis, adult-onset autoimmune diabetes, SLE, rheumatoid arthritis, psoriasis, and primary adrenal insufficiency.

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

Vogt-Koyanagi-Harada syndrome is a systemic multiorgan autoimmune disorder in which patients develop vitiligo of the head, neck, inner ear, and meninges. In addition to vitiligo, affected individuals develop severe uveitis, aseptic meningitis, dysacusis, tinnitus, poliosis, and alopecia.

<u>Management</u>



Vitiligo is most disfiguring on darker skin types and has a profound impact on the quality of life since patients experience stigmatization, isolation, and low self-esteem. Perform total body skin examination to assess:

- Type of vitiligo – nonsegmental (more common) or segmental
- Extent of disease
- Mucosal involvement
- Disease activity – stable or progressive
- Koebner phenomenon – new vitiligo lesions appear on areas affected by trauma
- Poliosis – depigmentation of hair follicles
- Use the "Rule of Nines" to estimate the total body surface area

Given the strong association of vitiligo with autoimmune thyroid disease, assess for signs and symptoms of thyroid dysfunction. Check thyroid stimulating hormone (TSH), anti-thyroperoxidase (TPO), and antithyroglobulin antibodies (TgAb). Consider additional autoantibodies if patient's history, family history, or physical examination suggests other autoimmune diseases, such as alopecia areata, psoriasis, type 1 diabetes, rheumatoid arthritis, psoriatic arthritis, inflammatory bowel disease, pernicious anemia, linear morphea, myasthenia gravis, discoid and systemic lupus erythematosus, and Sjögren's disease.

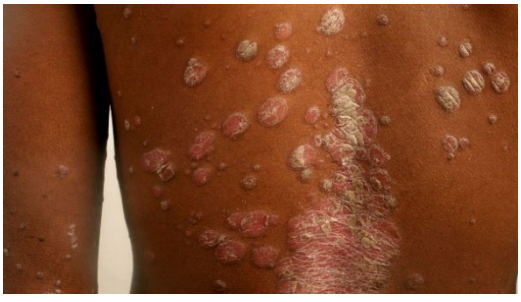
Although there is no cure for the disease, the available treatments may halt the progression of the disease and induce varying degrees of repigmentation, with acceptable cosmetic results in many cases. Combination therapies, such as phototherapy plus topical or oral corticosteroids, calcineurin inhibitors, or, less commonly, vitamin D analogues, appear to be more effective than single therapies.

Treatment includes broad-spectrum sunscreens, potent topical corticosteroids, topical calcineurin inhibitors (tacrolimus or pimecrolimus), narrow band ultraviolet (UV) B phototherapy, psoralen with UVA (PUVA) therapy, or total depigmentation for extensive disease. Refer to dermatology as clinically indicated.[35]

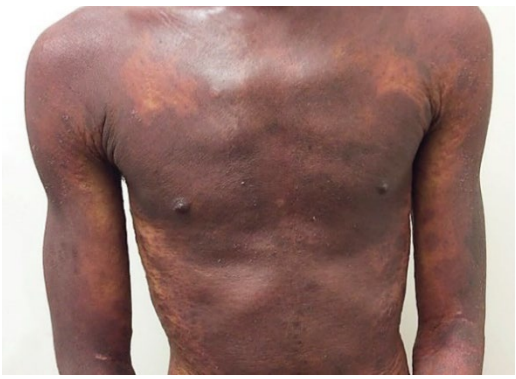
PSORIASIS

<u>Characteristics</u>

Psoriasis is a chronic, inflammatory, multisystem disease that affects up to 3.2% of the United States population. While skin involvement is the most prominent manifestation of this disease, recognition of psoriasis as a chronic, multisystem inflammatory disorder is imperative to optimize management and reduce comorbidities. The cutaneous manifestations are characterized by pruritic plaques covered with scales, particularly over the extensor surfaces, scalp, and lumbosacral region. On dark skin tones, a psoriasis plaque can range from erythematous to dark brown, purple, or gray. Plaque is thicker on darker skin tones with more scale. After psoriasis flare, areas of thickened skin will disappear, but discoloration remains on darker skin tones for months to a year. Nail changes in psoriasis are seen as pitting, oil spots, subungual hyperkeratosis, nail dystrophy, and anchylosis.

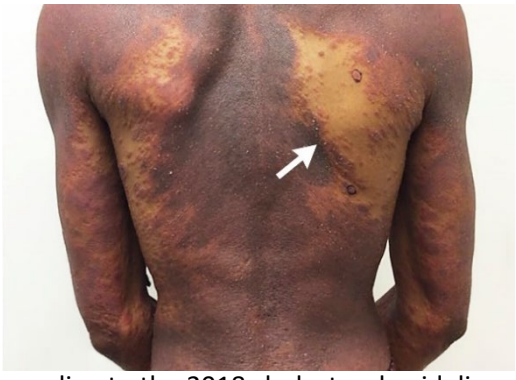


DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS



Psoriasis commonly affects the eye, including the eyelid, conjunctiva, and cornea, which gives rise to trichiasis, ectropion, conjunctivitis, and corneal dryness. The most common eye feature is blepharitis which can lead to cicatricial ectropion, madarosis, and trichiasis. Consider psoriasis in patients presenting with anterior uveitis.

Plaque psoriasis affects 85-90% of patients with psoriasis. Other morphologies of psoriasis include guttate, pustular, erythrodermic, inverse, and elephantine. Widespread pustular psoriasis and erythrodermic psoriasis have significant mortality rates.

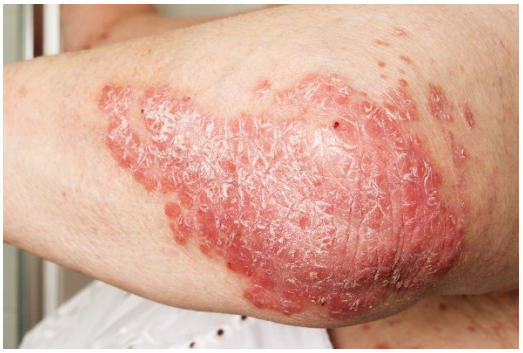


Psoriatic arthritis affects 30% of patients with psoriasis with an onset time of 10-11 years. It typically involves painful inflammation of the joints and connective tissue commonly affecting the joints of the fingers and toes. It leads to sausage-shaped swelling of the fingers and toes known as dactylitis. Psoriatic arthritis can also affect the hips, knees, and spine presenting as spondylitis and sacroiliac joints with sacroiliitis. The prevalence of psoriatic arthritis increased steadily with disease duration, reaching 20.5% among patients with a 30-year history of psoriasis. Patients with a higher total body surface area involvement are more likely to develop psoriatic arthritis.

Psoriasis is a risk-enhancing factor for atherosclerotic cardiovascular disease, according to the 2018 cholesterol guideline by the American Heart Association/American College of Cardiology. Psoriasis is associated with higher frequency of hypertension, diabetes, metabolic syndrome, inflammatory bowel disease, malignancy, serious infections, and other autoimmune disorders.

<u>Management</u>

After a comprehensive history, do a total body skin examination that includes examination of the scalp, nails, and anogenital skin. A skin biopsy may be performed if the diagnosis remains uncertain after the history and physical Routinely screen for signs and symptoms of psoriatic arthritis to facilitate early detection. Screening questions can include asking about the presence of joint pain, joint stiffness (particularly morning stiffness), and back pain (which patients may not recognize as a manifestation of joint pain). Consider appropriate laboratory testing (CBC with differential, CMP, urinalysis, ESR, CRP, rheumatoid factor, anti-cyclic citrullinated peptide, antinuclear antibody, and HLA-B27 testing) and imaging of affected joints and back for patients with signs and symptoms suspicious for psoriatic arthritis. Refer patients with psoriatic arthritis to rheumatology.



Also assess for other comorbid disease in patients with psoriasis:

- Inquire about personal and family history of coronary artery disease
- Screen for hypertension annually
- Screen Hgb A1c
- Screen lipid panel and calculate ASCVD risk, if clinically appropriate
- Screen for depression and anxiety
- Age-appropriate screening
- Annual skin examinations for patients on immunosuppressant therapy
- Screen for visual changes or ocular pain

Refer patients to dermatology based upon the severity of psoriasis, the total body surface area of skin affected by psoriasis, and the presence of signs or symptoms of comorbidities, such as psoriatic arthritis. Consider consultation with a rheumatologist as clinically indicated. A proactive approach to patient education and a consistent approach to routine

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

screening for signs and symptoms of psoriatic arthritis will facilitate the earliest possible detection. Patients are encouraged to notify their primary care providers if a musculoskeletal concern, such as morning joint stiffness or joint swelling, arises that cannot be explained.

Topical Treatment

- Daily skin care with emollient to reduce itching, desquamation, and total body surface area
- Topical corticosteroids for <4 weeks of plaque psoriasis not involving intertriginous areas and scalp psoriasis, using potency based on severity, plaque thickness, and plaque location
- Topical tacrolimus or pimecrolimus for <8 weeks of psoriasis involving the face and inverse psoriasis
- Long-term use of topical vitamin D analogues (up to 52 weeks), including calcipotriene or calcitriol, for the treatment of mild to moderate psoriasis
- Topical salicylic acid for 8-16 weeks for the treatment of mild to moderate psoriasis
- Coal tar preparations for the treatment of mild to moderate psoriasis
- Calcipotriene foam and calcipotriene plus betamethasone dipropionate gel for 4-12 weeks to treat mild to moderate scalp psoriasis
- Topical calcipotriene plus hydrocortisone for 8 weeks to treat facial psoriasis
- Morning high-potency topical corticosteroids plus evening topical vitamin D analogues for the treatment of psoriasis
- Vitamin D analogues twice daily on weekdays plus high-potency topical corticosteroids twice daily on weekends for maintenance treatment of psoriasis
- Mid- or high-potency topical corticosteroid plus tazarotene for 8-16 weeks for the treatment of mild to moderate psoriasis
- Combination of salicylic acid plus topical corticosteroids for the treatment of moderate to severe psoriasis (body surface area ≤20%)

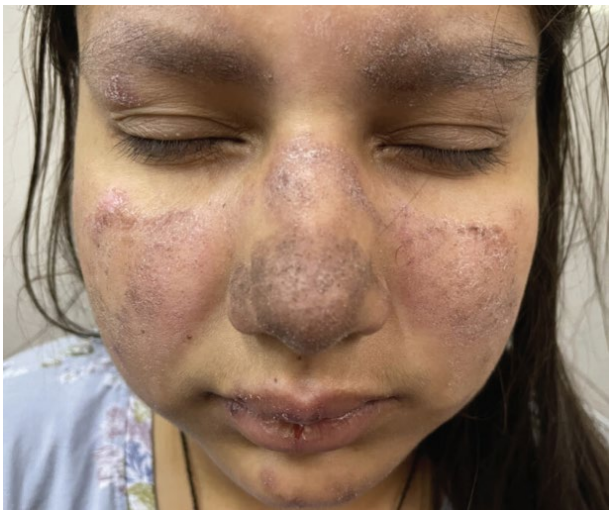
Topical Treatment in Combination with Systemic Therapy

- All topical corticosteroids can be used with biologic agents for the treatment of psoriasis
- Biologics, such as etanercept, adalimumab, infliximab, among other, have become the cornerstone of therapy for chronic moderate to severe plaque psoriasis as well as psoriatic arthritis

Refer to the Dyslipidemia Care Guide and the Hypertension Care Guide for more information about screening and cardiovascular disease risk management in patients with psoriasis. The target for blood pressure and lipid levels are based on risk calculated for psoriasis, which is a risk-enhancing factor for atherosclerotic cardiovascular disease.[36]

CUTANEOUS LUPUS ERYTHEMATOSUS AND SYSTEMIC LUPUS ERYTHEMATOSUS

Characteristics

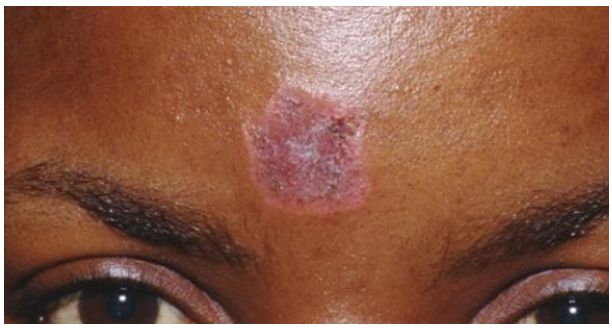


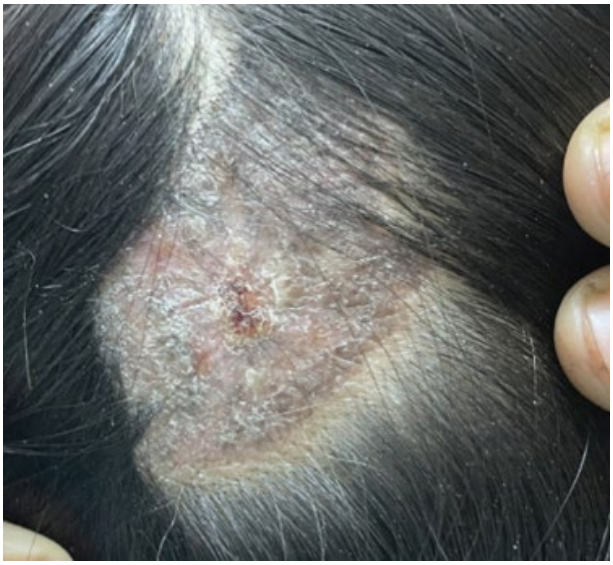
Cutaneous lupus erythematosus (CLE) is an autoimmune skin disease with a wide range of clinical presentations. Several subtypes exist including acute cutaneous lupus (ACLE), subacute cutaneous lupus (SCLE), and chronic cutaneous lupus (CCLE), as well as intermittent cutaneous lupus (ICLE) in some classification systems. The most common CCLE subtype being discoid lupus erythematosus (DLE). No universally accepted classification criteria exist for CLE.

While a minority of adult CLE patients will go on to develop systemic lupus erythematosus (SLE), this proportion is sizeable enough to highlight the need for CLE patients to have ongoing monitoring for the development of SLE. Disease severity, CLE subtype, autoantibodies (anti-dsDNA and anti-Smith), arthritis, and

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

high titers of ANA have been reported to be more commonly found in CLE patients progressing to SLE than those who have not.





For patients with SLE, skin is the second most frequently affected organ system with cutaneous manifestations occurring in 70% to 85% of individuals over the course of the disease and as a presenting symptom in 25% of patients. The 2019 European Alliance of Associations for Rheumatology (EULAR)/ACR classification criteria for SLE include positive antinuclear antibodies (ANA) followed by weighted criteria in 7 clinical and 3 immunologic domains. Patients accumulating > 10 points are classified as having SLE. Mucocutaneous is one of the 7 clinical realms, and includes alopecia (2 points), oral ulcers (2 points), ACLE or DLE (4 points), and ACLE (6 points).

SLE is a risk-enhancing factor for atherosclerotic cardiovascular disease, according to the 2018 cholesterol guideline by the American Heart Association/American College of Cardiology.

Management

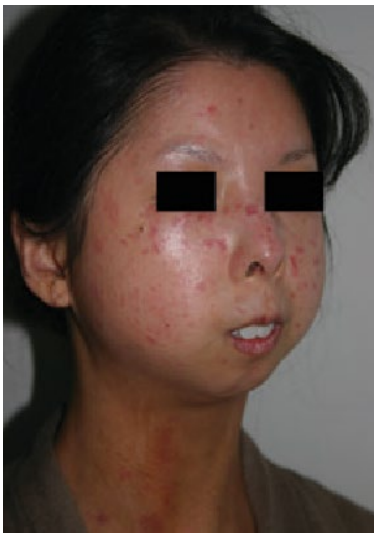
For patients in whom cutaneous findings are suspicious for CLE or SLE, refer to rheumatology for definitive diagnosis.

For patients with CLE, perform complete review of systems to identify any new systemic symptoms such as small joint pains, and thorough skin exams to check for worsening skin disease and presence of oral ulcers lasting more than two weeks. Laboratory tests including ANA and complete blood counts can be also ordered, with positive ANA titers being followed up with additional autoantibody tests including dsDNA and extractable nuclear antibody tests.

Comanage patients with CLE and SLE with specialists, like rheumatology. Also assess for other comorbid disease in patients with SLE, such as screening lipid panel and calculating ASCVD risk, since SLE is a risk-enhancing factor for atherosclerotic cardiovascular disease. Refer to the Dyslipidemia Care Guide and the Hypertension Care Guide for more information about screening and cardiovascular disease risk management, and the target for blood pressure and lipid levels are based on calculated risk.[37]

SYSTEMIC SCLEROSIS

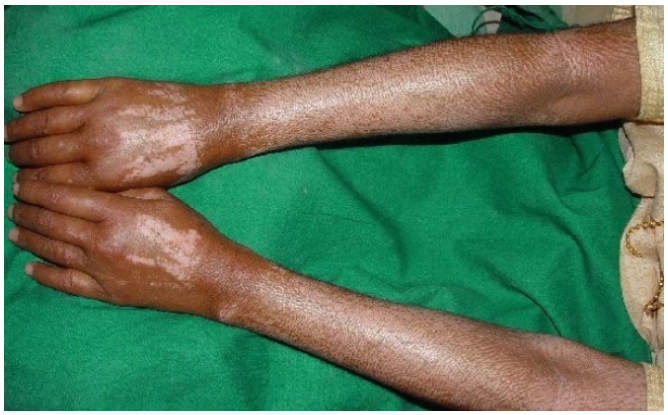
Characteristics



Patients with systemic sclerosis exhibit sclerotic changes of the skin but also have evidence of damage to visceral organs as a result of their autoimmune disease. Systemic sclerosis can be further subdivided into limited cutaneous (lcSSc) and diffuse cutaneous systemic sclerosis (dcSSc). Both limited and diffuse cutaneous systemic sclerosis patients have systemic disease characterized by Raynaud's phenomenon, the presence of autoantibodies, and internal organ involvement. Patients with limited cutaneous SSc usually have sclerosis only of the distal acral extremities and/or the face. They often present with a constellation of symptoms known as CREST syndrome (Calcinosis, Raynaud's, Esophageal Dysmotility, Sclerodactyly, and Telangiectasias). Patients with diffuse cutaneous SSc present with axial as well as acral sclerosis. They may have all of the above CREST manifestations as well but are also at high risk of developing significant pulmonary, renal, cardiac, musculoskeletal, and gastrointestinal manifestations of this disease.

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

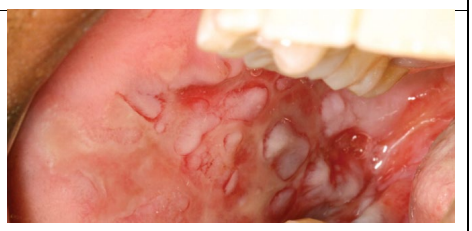
Skin thickening of the face results in reduced oral aperture. In some patients, cutaneous calcium deposits (calcinosis cutis) can be found on the hands and other parts of the acral appendages. Another cutaneous findings is hyper- and hypopigmentation known as a "salt & pepper rash". Furthermore, matted telangiectasias are common in systemic sclerosis.

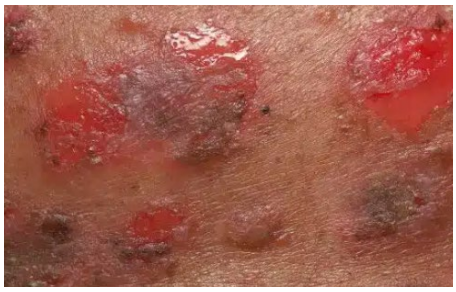


Management

Referral to rheumatology is recommended for comanagement. For patients in who systemic sclerosis is suspected, further workup and referrals to specialty care can be guided by symptoms to evaluation for specific organ involvement. Some areas to focus on include the GI tract (esophageal dysmotility, gastric antral vascular ectasia, small bowel bacterial overgrowth), lungs (interstitial lung disease, pulmonary hypertension), joints, muscles, and routine labs (e.g. anemia). Patients with early diffuse disease are also at risk for scleroderma renal crisis.[38]

PEMPHIGUS





Characteristics

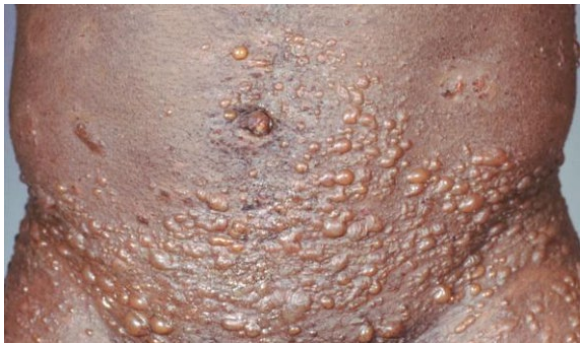
Pemphigus consists of a group of rare and severe autoimmune blistering diseases, which include pemphigus vulgaris, pemphigus foliaceus, IgA pemphigus, paraneoplastic pemphigus, and pemphigus vegetans. The most common form of pemphigus is pemphigus vulgaris.

Pemphigus is characterized by painful mucosal erosions and flaccid blisters that become erosive. 90% of patients have mucosal disease. Pemphigus vulgaris can develop at any age but commonly occurs between ages 40-60. Certain groups more prone to pemphigus vulgaris are people of eastern European Jewish and Mediterranean descent.

Management

Refer to dermatology and dental for comanagement. Treatment includes good wound care for affected skin, systemic corticosteroids, various immunosuppressants, rituximab, intravenous immunoglobulin, and plasmapheresis. Morbidity and mortality are significant despite treatment.[39]

PEMPHIGOID DISEASE

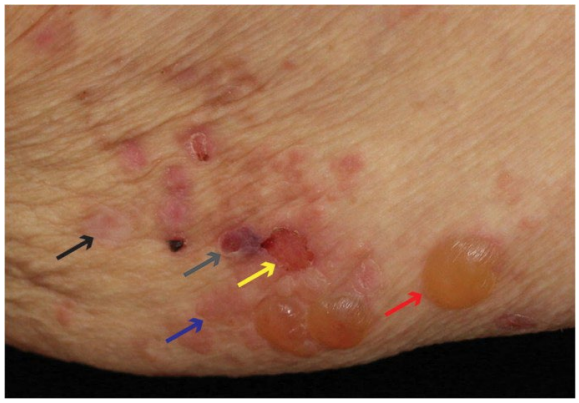


Characteristics

Pemphigoid diseases are a group of autoimmune blistering skin diseases. During an acute flare of disease, inflammatory skin lesions typically progressing from erythema through urticarial plaques to subepidermal blisters erosions erupt and, finally, completely resolve, thus illustrating that resolution of inflammation is continuously executed in pemphigoid disease patients and can be directly monitored on the skin. Seven different disease entities, namely bullous pemphigoid (BP), pemphigoid gestationis, mucous membrane pemphigoid, linear IgA disease, lichen planus pemphigoides, anti-p200 pemphigoid, and epidermolysis bullosa acquisita, belong to the group of pemphigoid disease entities.

DERMATOLOGIC CONDITIONS: INFLAMMATORY SKIN CONDITIONS

Bullous pemphigoid is the most common bullous disease and is characterized by large, tense subepidermal blisters, which are often very pruritic. Mucosal disease is rare. Bullous pemphigoid occurs most commonly in the elderly, usually >65 yo. Prognosis is influenced by age and general condition of the patient, not by extent of disease. This chronic disease usually exhibits a remitting-relapsing course.



Typically, inflammatory skin lesions in different stages of development coexist, including an emerging skin lesions in the shape of an urticarial plaque (blue arrow), a skin blister (red arrow), an erosion (yellow arrow), a resolving/healing erosion (grey arrow), and a resolved/healed skin lesions (black arrow) on the medial aspect of the upper arm of a BP patient during an acute flare.

Characteristics	Pemphigus Vulgaris	Bullous Pemphigoid
Age	40-60 yo	>65 yo
Symptoms	Painful	Itchy
Mucosa involved	Common	10-30%
Bullae	Flaccid, often ruptured, clear fluid	Tense, firm, fluid can be hemorrhagic
Staging of bullae	Monomorphic	Polymorphic (different stages)
Nikolsky sign	Positive	Negative
Prognosis	Poor	Favorable

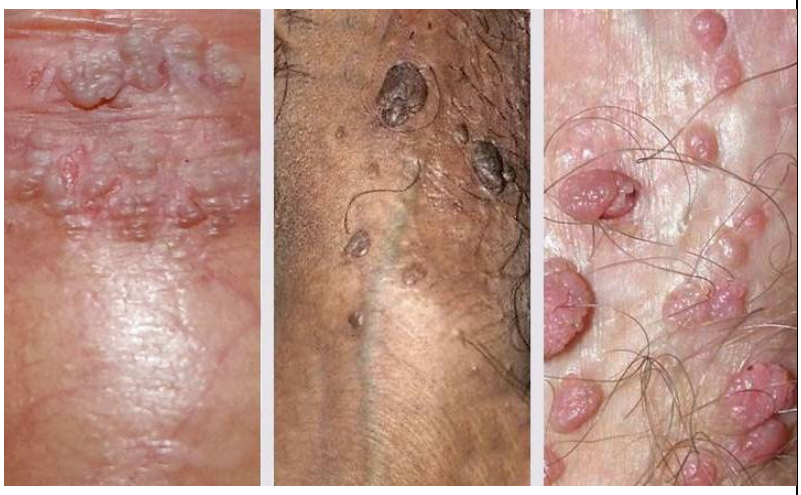
Management

Refer to dermatology for comanagement. Treatment of moderate and severe BP relies on the long-term use of systemic corticosteroids usually combined with potentially corticosteroid-sparing immunomodulators such as doxycycline and dapsone or immunosuppressants such as methotrexate, azathioprine, and mycophenolate. In severe or recalcitrant patients, adjuvant rituximab, immunoadsorption, or high-dose intravenous immunoglobulins can be employed.[39]

DERMATOLOGIC CONDITIONS: INFECTION

CONDYLOMATA ACUMINATA

Please refer to the Sexually Transmitted Infection Care Guide for more information. Anogenital warts are caused by *mucosal* human papillomavirus (HPV) types, most commonly HPV types 6 and 11, which are nononcogenic. However, HPV types 16, 18, 31, 33, and 34 are found in approximately 10% on anogenital warts and are associated with high-grade squamous intraepithelial lesions (HSIL), especially among patients living with HIV.



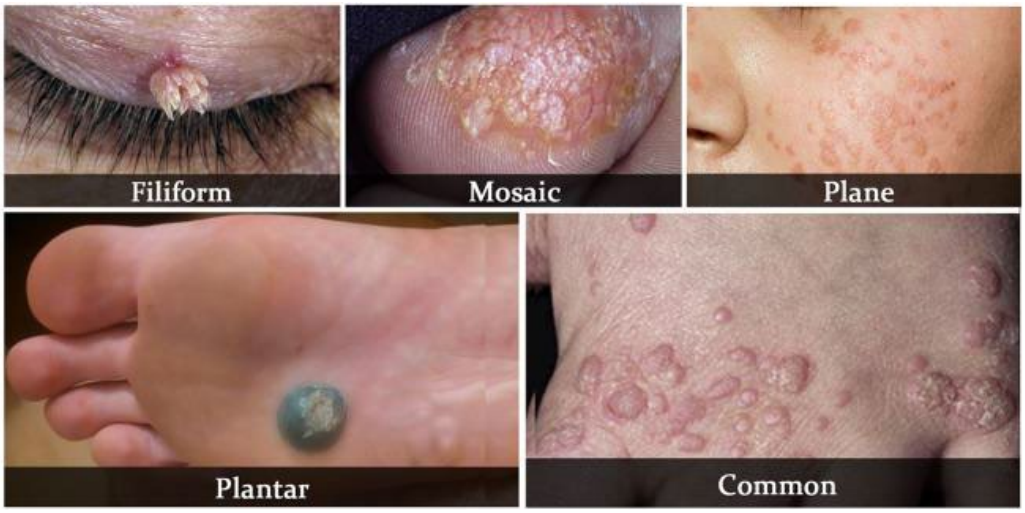
Most anogenital warts are asymptomatic, but some patients may present with pain or itching depending on location. Anogenital warts are skin-colored to brown flat, papular, or pedunculated lesions of various sizes.

Diagnosis is made by inspection and can be confirmed with biopsy if the diagnosis is uncertain or if malignancy is suspected.[40]

CUTANEOUS WARTS

Characteristics

Cutaneous warts are benign epithelial proliferations due to human papillomavirus (HPV) infection. *Cutaneous* HPV types cause common warts (verruca vulgaris), flat or plane warts (verruca plana), filiform warts (verruca filiformis), and plantar warts (verruca plantaris). Plantar warts that coalesce are described as mosaic warts. Cutaneous warts are skin-colored to brown papular, papillomatous, or pedunculated lesions of various sizes



Most cutaneous warts are asymptomatic, but hyperkeratotic warts and plantar warts can cause discomfort because of compression and friction. Large plantar warts can impair ambulation.

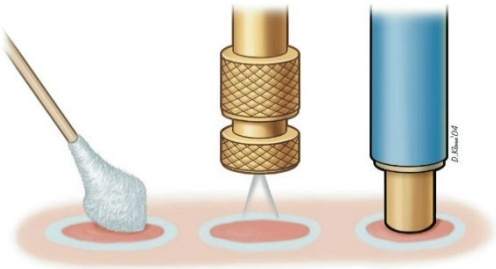
Management

Diagnosis is made by inspection and biopsy is rarely needed unless the diagnosis is uncertain.

Treatment depends on symptoms and patient preference. Nearly two-thirds of warts spontaneously disappear within 24 months, so consider observation if the patient is asymptomatic and the lesion is small. Cutaneous warts do not cause residual scarring when they spontaneously resolve. However, almost every type of topical treatment available has the potential to cause discomfort and/or scarring. Even though there are many treatments for warts, none is very effective, and recurrences are common with each of them.

DERMATOLOGIC CONDITIONS: INFECTION

Consider treatment if the patient has symptoms, the wart enlarges, or more warts spreads to other areas of the body. In most cases, topical agents are utilized first. Salicylic acid is often a first-line agent for the common wart with cure rates of 50-70%. Topical 5-fluorouracil under occlusion and topical imiquimod are also used for treatment. Other topical therapies, such as podophyllin and interferon, are not available on CCHCS Formulary.

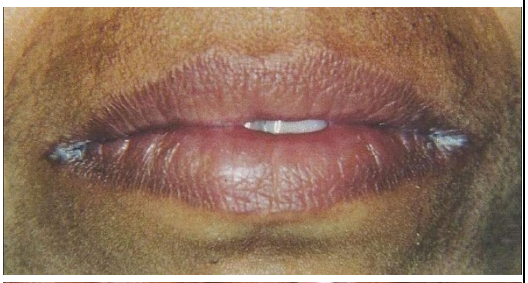


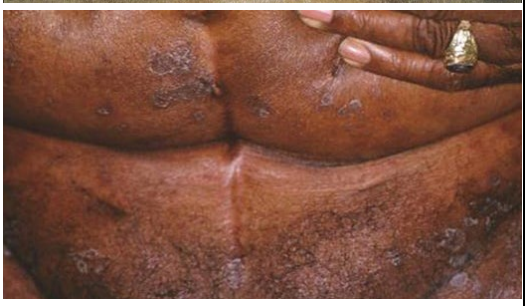
Most cutaneous warts require recurrent rounds of cryotherapy, every 3-4 weeks, for complete resolution. If cutaneous warts persist after 6 rounds of cryotherapy or grow and spread during appropriate therapy, consider referring to Dermatology for other surgical therapies, such as laser, electrodesiccation, and excision. Malignant change is rare with common warts but rarely one may encounter transformation to verrucous carcinoma, which is most common on the plantar surface.[40]

CUTANEOUS CANDIDIASIS

Characteristics

Cutaneous candidiasis is a common skin infection commonly affecting intertriginous skin. The primary intertriginous skin areas include the groin folds, axillae, and gluteal cleft. Body habitus due to overweight/obesity may contribute to additional intertriginous sites, such as inframammary skin and abdominal folds. Cutaneous candidiasis is also common in patients with diabetes, recent antibiotics use, steroid therapy, and chemotherapy.

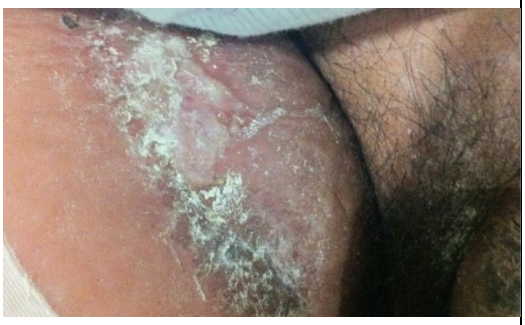




Patients present with plaques with peripheral pustules that range from beefy red on lighter colored skin to purple, brown, or gray on darker skin tones. *Candida* can infect the nails and corners of the mouth. Unlike tinea cruris, scrotal involvement can occur with candidiasis of the crural folds.

Cutaneous candidiasis may be inciting or exacerbating factors for some patients with intertrigo. To distinguish non-infectious intertrigo from bacterial intertrigo from candida intertrigo,

- Non-infectious intertrigo: moist with pruritic patches or plaques and peripheral scaling; can be painful with fissures or erosions
- Bacterial intertrigo: skin may be warm and edematous, suggestive of cellulitis with/without abscess
- Candida intertrigo: characteristic satellite papules and pustules often with a collarette of scale and a foul-smelling odor



Management

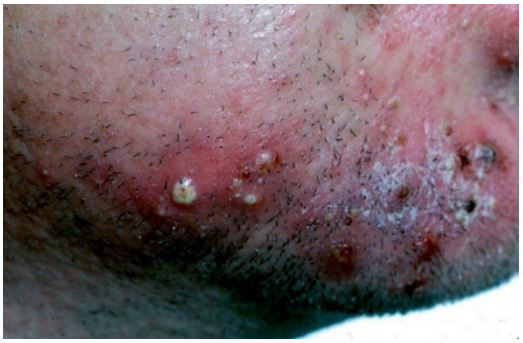
The diagnosis of cutaneous candidiasis can be made clinically based on the characteristic appearance and distribution of satellite papules and pustules. If clinically indicated, the diagnosis can be confirmed with a potassium hydroxide (KOH) preparation positive for pseudohyphae, which confirms *Candida* infection.

CCHCS Formulary **Topical Antifungals**	**Dose**
Clotrimazole 1% cream	Twice daily
Ketoconazole 2% cream or shampoo	Once daily
Miconazole 2% cream	Twice daily
Nystatin cream 100,000 units/gram	2-3 times daily

While non-infectious intertrigo is be treated with topical agents, such as zinc oxide, petrolatum, or dimethicone applied after cleaning and drying intertriginous skin, consider treating with a topical azole antifungal cream given how common cutaneous candidiasis is and the additional antibacterial and anti-inflammatory effects of these medications. Treat daily or twice daily with a topical azole for 2-4 weeks. Another topical treatment option is nystatin.[41]

DERMATOLOGIC CONDITIONS: INFECTION

TINEA BARBAE

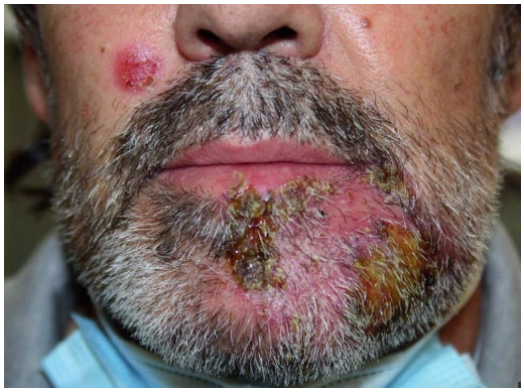


Characteristics

Tinea barbae is a rare dermatophytic infection. The classic inflammatory form of tinea barbae develops a kerion, which is a boggy, tender, weeping nodule or plaque with pustules and draining sinuses. Tinea barbae is typically restricted to the jawline, cheeks, or neck and rarely affects the upper lip. Patients may also present with malaise and fever. Kerion can get superinfected with cutaneous bacteria and develop regional lymphadenopathy.

Management

Consider eConsult to Dermatology for comanagement. For skin and soft tissue infections (SSTI), consider consult to Wound/SSTI Central Team. Tinea barbae can be diagnosed clinically based on a thorough history and physical examination. Occupational history and contact with domesticated animals will help narrow the differential diagnosis, particularly among patients with diabetes, local trauma, steroid use, and immunosuppression.



If diagnosis is uncertain, consider referral to Dermatology for direct microscopic examination of skin scrapings.

Specialized fungal culture and/or skin biopsy with histopathological examination can also be used to determine appropriate treatment, especially if the diagnosis is uncertain and refractory.

Treatment for tinea barbae is oral antifungal therapy for 4-6 weeks using one of the following CCHCS Formulary medications:

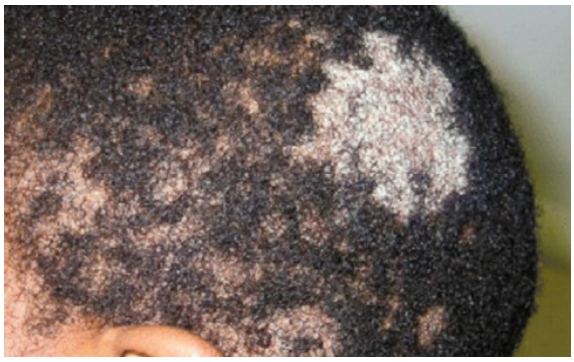
- Fluconazole 200 mg once daily
- Itraconazole 100 mg once daily[41]

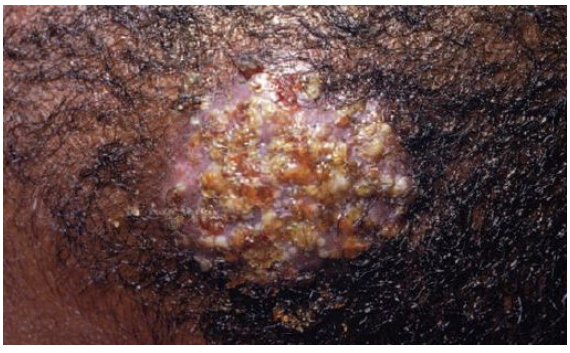
TINEA CAPITIS

Characteristics

Tinea capitis is a common dermatological disease, which affects children more than adults. However, immunosuppression can lead to impaired hair shaft growth and strength, which leads to easier colonization. Associated diseases include:

- Diabetes mellitus
- Prolonged steroid use
- Cancer
- Immunosuppressant medications
- Anemia





Typically, tinea capitis starts as inflamed papules that increase in size over time. Symptoms include itching, scaling, and alopecia. The presence of kerion, which is a boggy, tender, weeping nodule or plaque with pustules and draining sinuses, is associated with pus discharge and leads to scarring alopecia, which is permanent. Tinea capitis is often mistaken for severe seborrheic dermatitis, so consider tinea capitis for severe or refractory cases of seborrheic dermatitis or if there are areas of alopecia seen on physical examination.

DERMATOLOGIC CONDITIONS: INFECTION

Management

Consider eConsult to dermatology for suspected tinea capitis. For skin and soft tissue infections (SSTI), consider consult to Wound/SSTI Central Team. Consider a fungal culture swab, biopsy, or scraping from the scalp to confirm the causative fungal infection. Tinea capitis is treated with systemic antifungal medications for 4-8 weeks, and topical treatment is ineffective. Antifungal shampoos can be part of the treatment plan and often help in preventing spread, but this is not the mainstay of treatment.

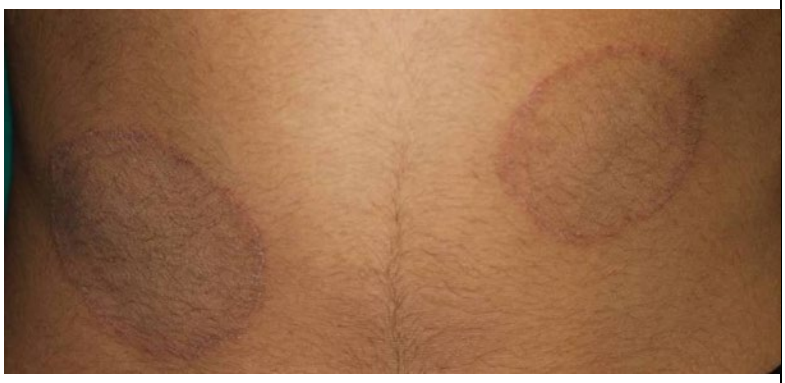
Often the oral antifungal therapies used are fluconazole, itraconazole, or griseofulvin. Presentations with kerion require adjunctive treatments with systemic steroids for a short period to reduce the inflammatory response and to lower the risk of permanent alopecia.[41]

TINEA CORPORIS

Characteristics

Tinea corporis is a common superficial fungal infection of body surfaces other than the feet, groin, face, scalp hair, or beard hair. Excessive heat, high relative humidity, and fitted clothing have correlations to more severe and frequent disease. Tinea corporis can also occur in outbreaks among athletes who have skin-to-skin contact

Vulnerable populations include patients with compromised immune systems, who may develop tinea corporis folliculitis, which invades the deep dermal layers. Other predisposing factors include underlying diseases such as diabetes mellitus, lymphomas, Cushing syndrome, excess sweating, or older age. There are also familial and genetic predispositions mediated by specific defects in innate and adaptive immunity.



Tinea corporis often begins as a circular or oval, erythematous or hyperpigmented, scaling patch or plaque. The skin lesion spreads centrifugally and develops central clearing. The result is a single or multiple pruritic annular (ring-shaped) plaques with a raised border and scaling. Extensive tinea corporis should prompt consideration of an underlying immune disorder, such as human immunodeficiency virus (HIV), or for diabetes.

Management

Tinea corporis usually responds well to topical antifungal medications, such as azoles, allylamines, butenafine, ciclopirox, and tolnaftate, applied 1-2 times daily for 1-3 weeks for clinical resolution. Topical nystatin is not effective treatment.

CCHCS Formulary **Topical Antifungals**	**Dose**
Clotrimazole 1% cream	Twice daily
Ketoconazole 2% cream or shampoo	Once daily
Miconazole 2% cream	Twice daily
Tolnaftate 1% cream	Twice daily

For patients with extensive skin involvement and patients who fail topical therapy, consider eConsult to dermatology for alternative diagnoses and comanagement. Oral antifungal treatment with fluconazole, itraconazole, terbinafine, or griseofulvin are used for severe and refractory tinea corporis. Patients are treated for 1-4 weeks depending on the oral antifungal regimen used, but longer courses can be used for some persistent cases. However, consider newer antifungal agents, such as posaconazole and voriconazole, for patients with tinea corporis resistant to standard oral antifungal therapies.[41]

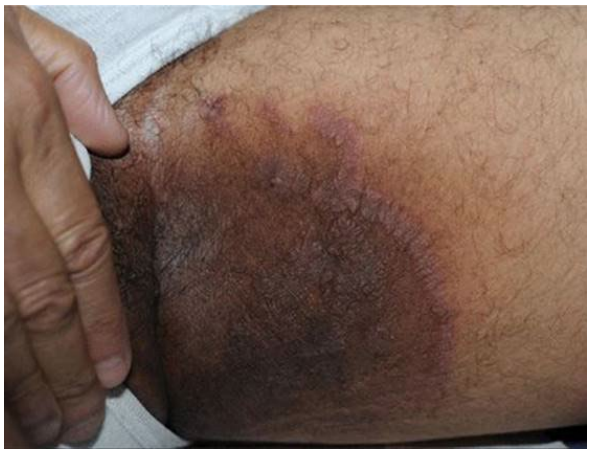
CCHCS Formulary **Oral Antifungals**	**Dose**	**Duration**
Fluconazole	150-200 mg once weekly	2-4 weeks
Itraconazole	200 mg daily	1 week

DERMATOLOGIC CONDITIONS: INFECTION

TINEA CRURIS

Characteristics

Tinea cruris often begins with an erythematous or hyperpigmented patch on the proximal medial thigh. The infection spreads centrifugally, with partial central clearing and a slightly elevated, erythematous or hyperpigmented, sharply demarcated border. Infection can involve the perineum and perianal areas, into the gluteal cleft, or onto the buttocks. Unlike cutaneous candidiasis with scrotal involvement, the scrotum is typically spared with tinea cruris.



Often, infection results from the spread of the dermatophyte infection from concomitant tinea pedis. Predisposing factors include copious sweating, obesity, diabetes, and immunodeficiency.

Management

Tinea cruris usually responds well to topical anti-fungal medications, such as azoles, allylamines, butenafine, ciclopirox, and tolnaftate, applied 1-2 times daily for 1-3 weeks for clinical resolution. Topical nystatin is not effective treatment.

CCHCS Formulary **Topical Antifungals**	**Dose**
Clotrimazole 1% cream	Twice daily
Ketoconazole 2% cream or shampoo	Once daily
Miconazole 2% cream	Twice daily
Tolnaftate 1% cream	Twice daily

For patients with extensive skin involvement and patients who fail topical therapy, consider eConsult to dermatology for alternative diagnoses and comanagement. Oral antifungal treatment with fluconazole, itraconazole, terbinafine, or griseofulvin are used for severe and refractory tinea cruris. Patients are treated for 1-4 weeks depending on the oral antifungal regimen used, but longer courses can be used for some persistent cases. However, consider newer anti-fungal agents, such as posaconazole and voriconazole, for patients with tinea cruris resistant to standard oral anti-fungal therapies.

CCHCS Formulary **Oral Antifungals**	**Dose**	**Duration**
Fluconazole	150-200 mg once weekly	2-4 weeks
Itraconazole	200 mg daily	1 week

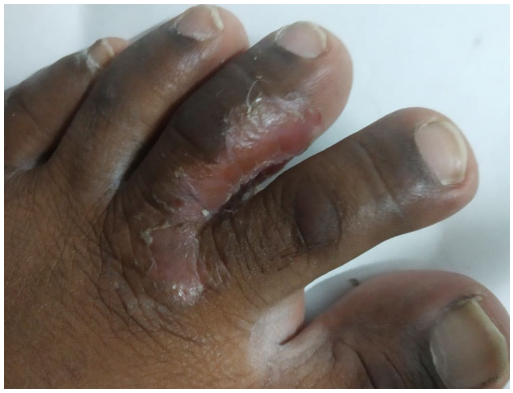
Recurrence of tinea cruris is common. Concomitant tinea pedis should be treated to reduce risk for recurrence. Treatment of onychomycosis can also reduce recurrences. Other interventions include daily use of desiccant powders or drying lotions in the inguinal area and avoidance of tight-fitting clothing and noncotton underwear.[41]

TINEA PEDIS

Characteristics

Tinea pedis, also known as athlete's foot, can present as:

- Interdigital tinea pedis: macerated interdigital skin with pruritic erosions or scales between the toes, which can lead to painful fissures
- Vesiculobullous tinea pedis: pruritic, sometimes painful, vesicular or bullous eruption, often involving the medial foot
- Hyperkeratotic tinea pedis: diffuse, hyperkeratotic eruption involving the soles and medial and lateral surfaces of the feet



Secondary bacterial infection is an infrequent presentation. Tinea pedis can occur in association with onychomycosis, tinea cruris, or tinea manuum.

DERMATOLOGIC CONDITIONS: INFECTION

Management

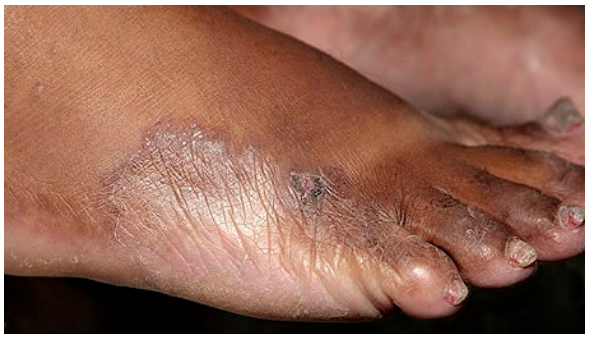
Advise all patients with tinea pedis to keep their feet and socks clean and dry.

Tinea pedis usually responds well to topical anti-fungal medications, such as azoles, allylamines, butenafine, ciclopirox, and tolnaftate, applied 1-2 times daily for 1-4 weeks for clinical resolution. Topical nystatin is not effective treatment.

CCHCS Formulary **Topical Antifungals**	**Dose**
Clotrimazole 1% cream	Twice daily
Ketoconazole 2% cream or shampoo	Once daily
Miconazole 2% cream	Twice daily
Tolnaftate 1% cream	Twice daily

Consider using terbinafine 1% cream applied to interdigital tinea pedis for a week given the high cure rate of this regimen. For patients with interdigital maceration or vesiculation, place gauze or cotton between the toes.

Consider adding a topical keratolytic agent, such as salicylic acid, to accelerate resolution of scale for patients with hyperkeratotic tinea pedis.

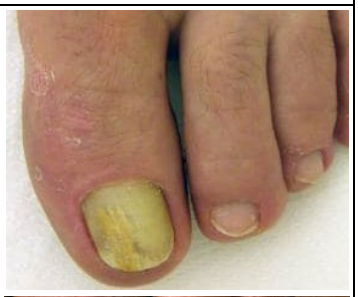


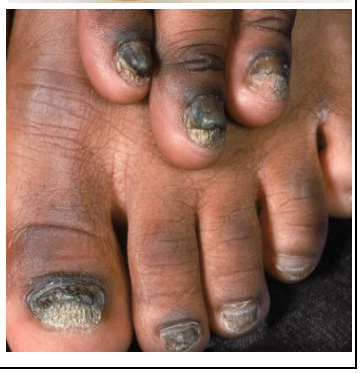
For patients with extensive skin involvement and patients who fail topical therapy, consider eConsult to dermatology for alternative diagnoses and comanagement. Oral antifungal treatment with fluconazole, itraconazole, terbinafine, or griseofulvin are used for severe and refractory tinea pedis. Patients are treated for 1-4 weeks depending on the oral antifungal regimen used, but longer courses can be used for some persistent cases.

CCHCS Formulary **Oral Antifungals**	**Dose**	**Duration**
Fluconazole	150-200 mg once weekly	2-4 weeks
Itraconazole	200 mg daily	1 week

Recurrence of tinea pedis can be prevented by wearing socks with wick-away material, using desiccating foot powder, avoidance of occlusive footwear, and treating hyperhidrosis. Treatment of onychomycosis can also reduce recurrences. Refer to the Chronic Wound Management Care Guide for open wounds, as well as the Foot Care Guide for additional information.[41]

ONYCHOMYCOSIS





Characteristics

Patients with onychomycosis can present with nail discoloration, subungual hyperkeratosis, onycholysis, and nail plate destruction. Onychomycosis is caused by direct contact with environmental fungus and direct spread, as is the case for tinea pedis. The most common (60-70%) type of onychomycosis is caused by dermatophyte infection, also called tinea unguium. Chronic paronychia is a comorbid condition found with *Candida* onychomycosis.

Most onychomycosis presents as subungual onychomycosis but can progress to total dystrophic onychomycosis, which is the complete destruction of the nail plate that results in a thickened and ridged nail bed covered with keratotic debris. This leads to nail disfigurement, pain, and a reservoir for recurrence of tinea corporis, tinea cruris, and tinea pedis.

Management

Despite the availability of multiple therapies, treatment failure and onychomycosis recurrence are common. Given the broad differential diagnosis of nail dystrophy, consider

DERMATOLOGIC CONDITIONS: INFECTION

confirming the presence of fungi prior to treatment with fungal culture. Fungal cultures also identify the type of fungus present and confirm fungal viability.

Most cases of onychomycosis are cosmetic and do not require treatment. Advise all patients with onychomycosis to keep their feet and socks clean and dry.

Consider offering treatment of onychomycosis to the following:

- Patients with a history of cellulitis of the lower extremity with ipsilateral toenail onychomycosis
- Patients with diabetes and toenail onychomycosis who have additional risk factors for cellulitis, such as prior cellulitis, venous insufficiency, or edema
- Patients who are experiencing discomfort or pain associated with infected nails [2-4]
- Patients who are immunosuppressed

Patients can be treated empirically for dermatophyte infection, which is the most common type of onychomycosis, using oral antifungal therapy. Topical anti-fungal agents developed for cutaneous fungal infections generally are poorly effective for onychomycosis because of poor penetration of the nail plate. The preferred first-line oral antifungal is terbinafine, but this is not available on CCHCS Formulary. Itraconazole is an alternative (second-line) systemic treatment. The potential for drug-drug interactions is higher for itraconazole than terbinafine, but itraconazole treats onychomycosis due to dermatophytes, nondermatophytes, and yeast.

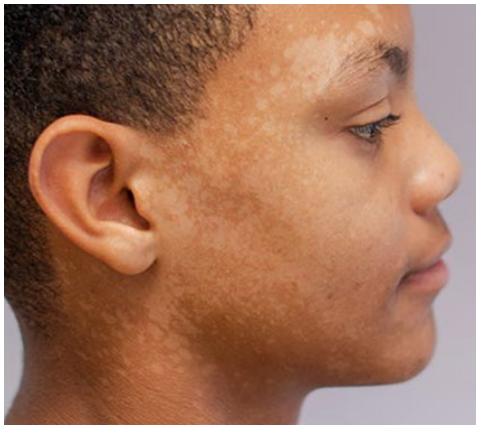
Oral Antifungals	Dose	Duration
Terbinafine	250 mg daily	6 weeks for fingernail onychomycosis 12 weeks for toenail onychomycosis
Itraconazole (CCHCS Formulary)	200 mg daily	6 weeks for fingernail onychomycosis 12 weeks for toenail onychomycosis

For patients who forgo antifungal treatment or as an adjunct to antifungal treatment, removal of hyperkeratotic nail debris can improve pain with ambulation and facilitate nail trimming of very thick, dystrophic nails. Consider nail removal and debridement for severe cases. Removal of hyperkeratotic nail debris can also be accomplished with topical urea, a keratolytic agent, under occlusion overnight. Reapplication of topical urea without occlusion on an as-needed basis can be used to maintain improvement. Treatment without occlusion also is often sufficient for reducing nail hyperkeratosis in patients with less severe nail thickening. Refer to the Foot Care Guide for additional information.[42]

TINEA VERSICOLOR

Characteristics

Tinea versicolor, also known as pityriasis versicolor, is a common superficial fungal infection that presents with hypopigmented, hyperpigmented, or erythematous macules on the face, trunk, and proximal upper extremities. In patients with darkly pigmented skin, hyperpigmented tinea versicolor often presents as dark brown to gray-black lesions, and in patients with lightly pigmented skin, hyperpigmented tinea versicolor is often light to medium brown.

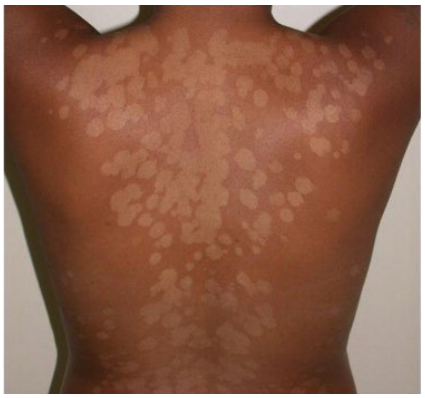


Individual lesions are typically small, oval or round but coalesce into larger patches or thin plaques. A fine scale is often present, which becomes more apparent when the lesion is scraped for microscopy. Patients are asymptomatic or may have mild pruritus.

DERMATOLOGIC CONDITIONS: INFECTION

Management

Diagnosis can be based upon the physical examination. For most patient, topical therapy is preferred using topical azole antifungal medication, topical selenium sulfide, or topical terbinafine. Caused by *Malassezia*, tinea versicolor responds well to therapy, but recurrence is common



If topical therapy is ineffective, consider using a different topical therapy or starting oral antifungal therapy. However, persistent hypopigmentation or hyperpigmentation is not a reliable indicator of treatment failure, since skin discoloration can persist for weeks to months after successful treatment. The presence of the characteristic fine scale of tinea versicolor is a clinical sign that suggests active infection, or confirmation can be made with potassium hydroxide (KOH) preparation. Consider immunodeficiency or alternative diagnosis for extensive disease and resistance to topical therapy.[41]

CCHCS Formulary **Topical Therapy**	**Dose**	**Duration**
Ketoconazole 2% shampoo	Once daily x 5 minutes, then wash	3 days
Ketoconazole 2% cream	Once daily	2 weeks
Clotrimazole 1% cream	Once daily	2 weeks
Miconazole 2% cream	Once daily	2 weeks
Selenium sulfide 2.5% shampoo	Once daily x 10 minutes, then wash	1 week

CCHCS Formulary **Oral Antifungals**	**Dose**	**Duration**
Fluconazole	300 mg once weekly	2 weeks
Itraconazole	200 mg daily	5 days

DERMATOLOGIC CONDITIONS: DISORDERS OF HAIR

ALOPECIA AREATA

Characteristics

Alopecia areata is a chronic, relapsing autoimmune disorder that causes nonscarring hair loss in distinct patches on the scalp as well as other parts of the body. Severity ranges from small patches of alopecia to the complete loss of scalp, eyebrow, eyelash, and body hair.

Alopecia areata has "exclamation mark" hair that tapers towards to tip. There is regrowing vellus and white hairs. Trichotillomania has broken hair of different lengths as well as areas of no hair.

Management

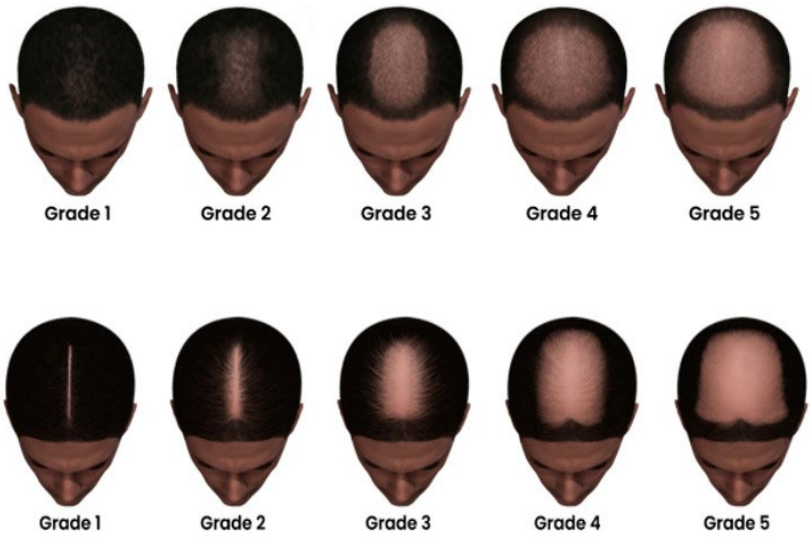
Up to 50% of patients who present with patchy alopecia areata experience spontaneous hair regrowth within one year. Response to treatment varies and recurrence can occur.

Because of the association between alopecia areata and autoimmune thyroid disease and the relatively high frequency of thyroid disease in the general population, an assessment for thyroid disease is an important aspect of management. For patients with clinical signs or symptoms suggestive of other autoimmune diseases, consider targeted assessments of suspected disorders with the following laboratory tests: thyroid stimulating hormone (TSH), iron studies with ferritin, serum zinc, 25-hydroxyvitamin D, and/or serum testosterone. In the absence of such features, routine testing is not recommended.

ANDROGENETIC ALOPECIA

Characteristics

Androgenetic alopecia occurs in characteristic male and female patterns and characteristically manifests with slow, progressive nonscarring hair thinning. The condition is characterized by the progressive loss of terminal hairs over the frontal and vertex regions of the scalp, resulting in a visible reduction in hair density. Unlike male pattern hair loss, the frontal hairline is often spared in female pattern hair loss.



Management

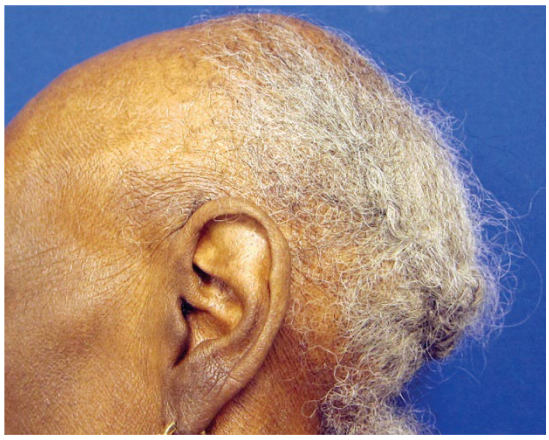
Androgenetic alopecia is cosmetic and does not require treatment. Additionally, no treatments for androgenetic alopecia are curative, so long-term continuation of pharmacologic therapy is necessary to maintain benefit.

However, in some instances, androgenetic alopecia can be associated with other comorbidities that can potentially contribute to adverse health outcomes. For example, early hair loss and hair loss involving the vertex is associated with cardiovascular disease. Additionally, female pattern hair loss is associated with polycystic ovarian syndrome, so treating the underlying, hyperandrogenic state is important for improving female pattern hair loss associated with hyperandrogenism.

Lastly, management of comorbid inflammatory scalp conditions, such as seborrheic dermatitis, minimizes hair loss.

DERMATOLOGIC CONDITIONS: DISORDERS OF HAIR

TRACTION ALOPECIA



Characteristics

Although no biochemical differences of hair have been found among persons of different ethnicities, there are notable differences in the shape of the hair follicle, leading to differences in texture, fragility, and manageability of hair. Persons of African descent have the greatest degree of curl, and mechanical fragility increases with higher degrees of curl. The hair is drier with less sebum production and can be difficult to comb in its natural state, resulting in many distinct hair care practices. Styles such as braids and weaves may increase traction alopecia at the frontal and temporal hair lines if there is excess pull on the hair.

Management

Because excess hair washing can lead to breakage, shampoo hair every 1-2 weeks to prevent buildup of styling product, irritant dermatitis, and seborrheic dermatitis. Conditioning helps prevent hair fragility. Emollients can improve manageability, but this can lead to acne, irritant contact dermatitis, and seborrheic dermatitis. Instruct patients to apply emollients only to hair and limit contact with the face. Caution patients about the tightness and pull on the natural hair.

For patients with signs of inflammation, topical corticosteroid therapy can be applied twice daily to the entire affected area. Consider prescribing oral tetracycline antibiotics, such as tetracycline 500 mg twice daily or doxycycline 50-100 mg twice daily, for patients with signs of inflammation, especially when pustules are present. A typical treatment course is one month or until resolution of the folliculitis.

TELOGEN EFFLUVIUM

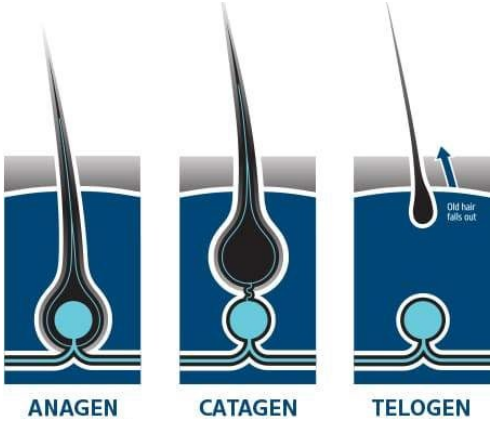
Characteristics

Telogen effluvium is a form of diffuse, nonscarring hair loss that presents as a transient or chronic loss of hair due to premature shedding of hair. A wide variety of factors have been linked to telogen effluvium, such as COVID-19 infection, major surgery, serious illness, childbirth, protein or caloric malnutrition, drugs, and severe emotional distress.

Management

Through history and physical exam determine the cause of telogen effluvium:

- Date of onset, duration, and potential triggers inciting hair loss
- Estimated amount of hair lost daily
- Recent past medical history, such as surgeries, weight loss, pregnancy, psychological stressors
- History of medication or toxic exposure
- Family history of hair loss
- Scalp assessment of scale, inflammation, or scarring
- Intact or broken shafts of hair



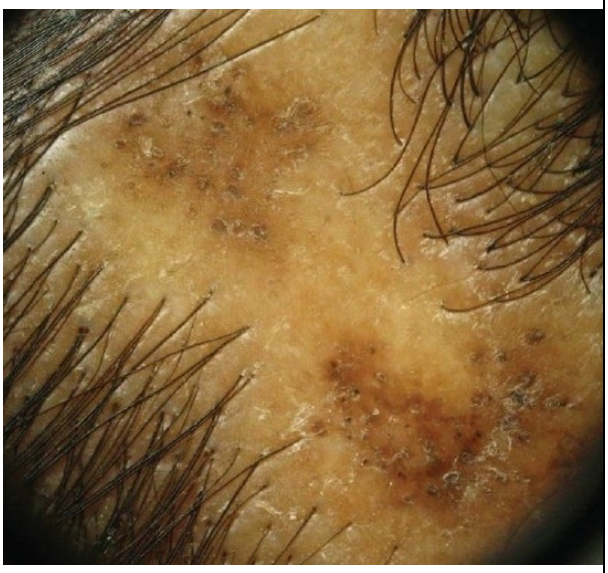
Consider the following lab tests to assess for underlying causes:

- Complete blood count (CBC) with red blood cell indices
- Complete metabolic panel (CMP)
- Thyroid-stimulating hormone (TSH)
- Ferritin
- 25-hydroxyvitamin D levels

Although spontaneous improvement is expected for patients with telogen effluvium related to an isolated event (eg, childbirth), patients with telogen effluvium related to a persisting insult should have the cause eliminated or treated when feasible, such as iron supplementation for iron deficiency anemia.

DERMATOLOGIC CONDITIONS: DISORDERS OF HAIR

CICATRICIAL (SCARRING) ALOPECIA



Characteristics

Primary cicatricial alopecies are inflammatory disorders of the scalp that lead to permanent hair. The primary cicatricial alopecias are subdivided by the type of inflammation detected on histologic examination. The three major classes are lymphocytic primary cicatricial alopecias, neutrophilic primary cicatricial alopecias, and mixed primary cicatricial alopecias. The most common primary cicatricial alopecias are

- Lichen planopilaris: This type of lichen planus is the most common primary cicatricial alopecia, mostly affecting patients over 50 who are assigned female at birth (AFAB).
- Central centrifugal cicatricial alopecia (CCCA): CCCA is the most common type of hair loss in Black patients AFAB. It usually occurs around age 30 as a bald patch at the vertex that spreads outward.
- Chronic cutaneous lupus erythematosus (CCLE): CCLE can also cause cicatricial alopecia

Secondary cicatricial alopecia is a side effect of injury or damage to skin. Hair loss might result from radiation therapy, burns, infections, or tumors.

Clinical symptoms such as burning, itching, and scalp tenderness are associated with the primary cicatricial alopecias but can also be present in alopecia areata, a nonscarring alopecia. Therefore, it is important to determine if the patient is experiencing any of these symptoms to narrow the differential diagnosis.

Absence of follicular ostia is one of the cardinal signs of cicatricial alopecia. Cicatricial alopecia can also present erythema or hyperpigmentation, scaling or crust under Wood's lamp, perifollicular hyperkeratosis, atrophy, or pustules. Irregularly paced hair shafts and hair tufting are additional features of scarring.

Management

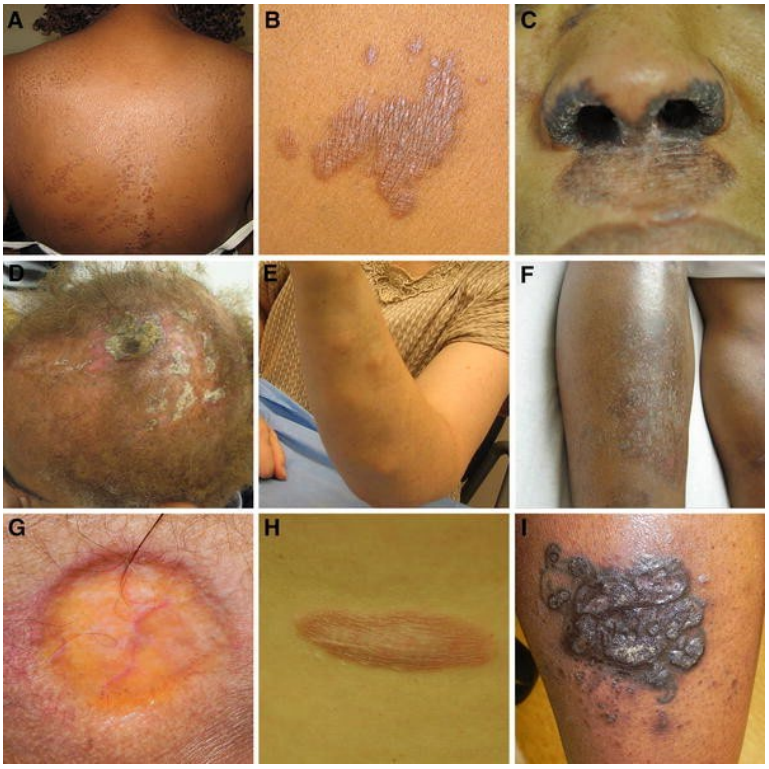
Biopsy is needed for a diagnosis, so consider referral to dermatology.

DERMATOLOGIC CONDITIONS: DERMATOLOGIC MANIFESTATIONS OF SYSTEMIC DISEASE

SARCOIDOSIS

Characteristics

Sarcoidosis is a multisystem, granulomatous disease of the lungs, bones, central nervous system, lymph nodes, eyes, and skin. Skin disease, affecting 25-35% of patients.



Lupus pernio is the most distinctive cutaneous lesion of sarcoidosis, which presents on the nose, middle face, and cheeks as red to violaceous, indurated plaques with infrequent superficial scaling leaving behind telangiectatic scars. Since there is a close association between upper respiratory tract sarcoidosis and lupus pernio, early diagnosis and aggressive therapy is needed.

Erythema nodosum, an acute, painful panniculitis that usually affects the shins, is the most common nonspecific cutaneous manifestation of sarcoidosis, but sarcoidosis is also associated with other nonspecific cutaneous findings, such as erythema multiforme, prurigo nodularis, and calcinosis cutis.

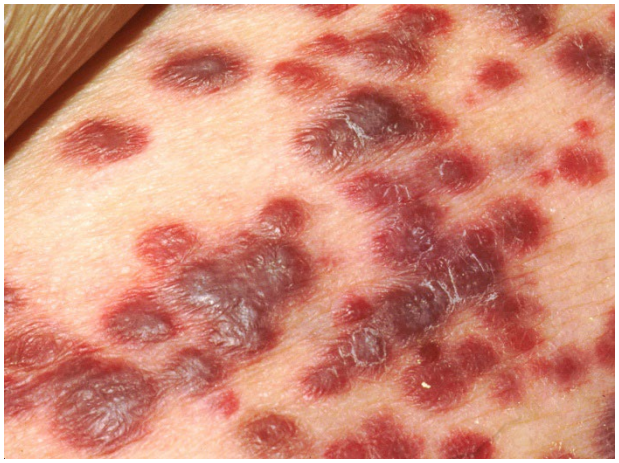
Management

For suspected sarcoidosis, refer the patient to pulmonology for a definitive diagnosis.[44]

HIV

Characteristics

Dermatological disorders are among the most prevalent manifestations of HIV, ranging from acute primary HIV rash and infections (herpes simplex, molluscum contagiosum, scabies) to cutaneous malignancies (Kaposi's sarcoma, lymphoma, nonmelanoma skin cancers) and antiretroviral therapy (ART)-associated drug eruptions. Other prevalent dermatoses seen among patients living with HIV who have lower CD4 counts include papular pruritic eruption, eosinophilic folliculitis, prurigo nodularis.



Common dermatologic disorders are reported more frequently in patients living with HIV than the general population. These dermatologic conditions can be more severe among patients living with HIV but can regress with ART.

- Seborrheic dermatitis
  - Affects 30-80% of patients living with HIV vs 1-3% of general population
  - Thicker, yellowish scale
  - More frequent relapses
- Psoriasis
  - More severe with nail involvement
  - More likely to develop severe psoriasis, treatment-refractory disease, and psoriatic arthritis
  - Typically present with guttate, inverse, and erythrodermic psoriasis

DERMATOLOGIC CONDITIONS: DERMATOLOGIC MANIFESTATIONS OF SYSTEMIC DISEASE

- Atopic dermatitis
  - Often new adult-onset and generalized
  - Associated with lymphadenopathy and constitutional symptoms

Management

Comanage patients with HIV Central Team. Refer to HIV Care Guide.[45]

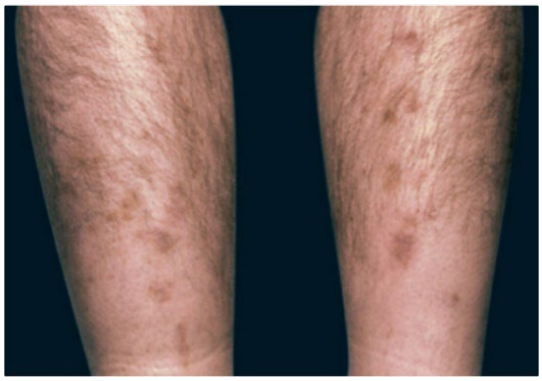
DIABETES

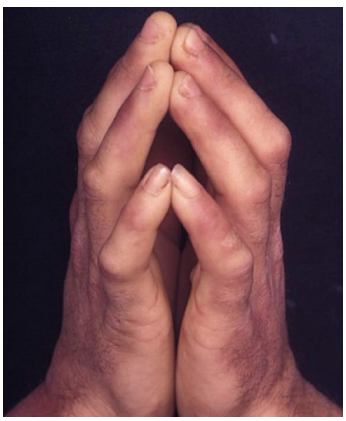
Characteristics

Acanthosis nigricans is most commonly associated with diabetes and insulin resistance. Since the vast majority of cases are associated with insulin resistance and/or obesity, screening for diabetes and measuring glycosylated hemoglobin is recommended.[17]

Diabetes dermopathy presents with rounded, dull, red papules that progressively evolve over one-to-two weeks into well-circumscribed, atrophic, brown macules with a fine scale. Normally after about eighteen to twenty-four months, lesions dissipate and leave behind an area of concavity and hyperpigmentation. At any time, different lesions can present at different stages of evolution. The lesions are normally distributed bilaterally and localized over bony prominences. The pretibial area is most commonly involved, although other bony prominences such as the forearms, lateral malleoli or thighs may also be involved.

Diabetes foot syndrome (DFS) encompasses the neuropathic and vasculopathic complications that develop in the feet of patients with diabetes. Although preventable, DFS is a significant cause of morbidity, mortality, hospitalization, and reduction in quality of life of patients with diabetes. DFS presents initially with callosities and dry skin related to diabetic neuropathy. In later stages, chronic ulcers and a variety of other malformations of the feet develop. Between 15% and 25% of patients with diabetes will develop ulcers. Ulcers may be neuropathic, ischemic, or mixed. The most common type of ulcers are neuropathic ulcers, a painless ulceration resulting from peripheral neuropathy. Ulcers associated with peripheral vascular ischemia are painful but less common. Ulcers tend to occur in areas prone to trauma, classically presenting at the site of calluses or over bony prominences. It is common for ulcers to occur on the toes, forefoot, and ankles.





Scleroderma-like skin changes are found in 10-50% of patients with diabetes. These findings occur more commonly in those with type 1 diabetes and in those with longstanding disease. Signs develop slowly and present with painless, indurated, occasionally waxy appearing, thickened skin. Patients are often asymptomatic, but patients can have a reduction in sensation and pain. In patients with scleroderma-like skin changes the acral areas are involved symmetrically, specifically the dorsum of the fingers (sclerodactyly), proximal interphalangeal, and metacarpophalangeal joints. Severe disease may extend centrally from the hands to the arms or back. Over time, limited joint mobility (LJM) leading to progressive flexed contractures and hindered joint extension. Patients may present with an inability to flushly press the palmar surfaces of each of their hands together (“prayer sign”) or against the surface of a table when their forearms are perpendicular to the surface of the table (“tabletop sign”).

DERMATOLOGIC CONDITIONS: DERMATOLOGIC MANIFESTATIONS OF SYSTEMIC DISEASE

Other rarer skin manifestations of diabetes include scleroderma diabetocorum, necrobiosis lipoidica, and bullous diabeticorum. Some nonspecific dermatologic signs and symptoms of diabetes also include ichthyosiform changes of the skin, xerosis leading to diabetes-associated pruritus, keratosis pilaris, eruptive xanthomas at lower triglyceride levels, and acrochordons, to name a few.

Management
Refer to Diabetes Care Guide, Foot Care Guide, and Chronic Wound Management Care Guide.

SUBSTANCE USE DISORDER

Characteristics

Dermatologic manifestations of cocaine use include cuts and burns on the lips from broken pipes, madarosis from hot steam, and palmar and digital hyperkeratosis. Cocaine use has been linked to vasculitis and formication, the tactile hallucination of insects crawling under the skin, leading to skin picking and excoriations. The vasoconstrictive effect of cocaine causes local ischemia, leading to necrosis of the intranasal mucosa. This can even affect deep osteocartilaginous structures and cause anatomic cocaine-induced midline destructive lesions

Patients who use heroin can develop urticaria, intense pruritus on the face and genital region, penile ulcers after injection into the dorsal penile vein, and necrotizing cellulitis of the scrotum after injection into the femoral artery.

Methamphetamine use has been shown to produce xerosis, pruritus, intense body odor, premature skin aging, hyperhidrosis, acne, and lichenoid drug eruptions.

Shared paraphernalia can also transmit infectious diseases with cutaneous manifestations, such as HPV causing intranasal “snorters’ warts”.

Nasal Route	Inhaled Route	Intravenous Route
Intranasal viral warts	Burns on hand, face, lips	Scars (track marks, skin popping)
Intranasal and perinasal crusts	Viral warts on hands	Hypertrophic scars and keloids
Ulcerations	Madarosis	Foreign body granulomas
Necrosis	Mucositis	Skin and soft tissue infections

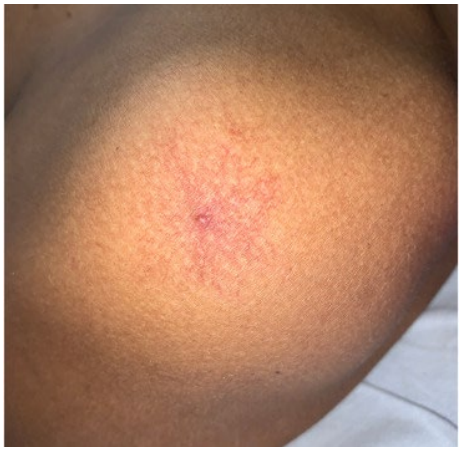
Management
Refer to Substance Use Disorder Care Guide.[47]

HEPATITIS C

Characteristics

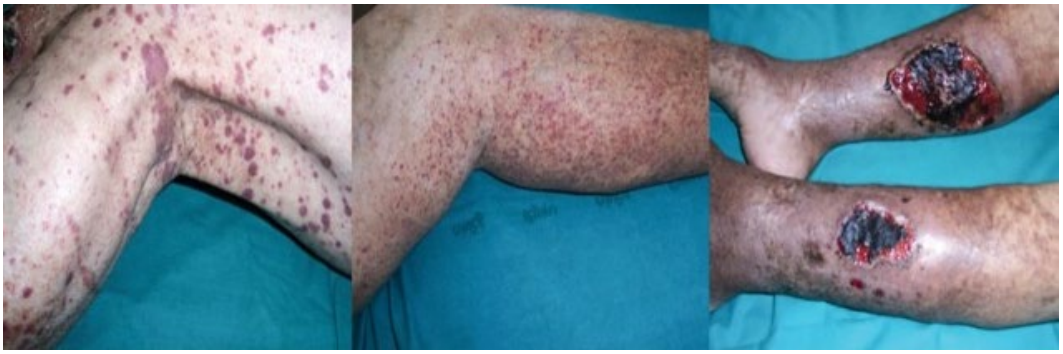
Hepatitis C virus causes several dermatologic extrahepatic manifestations (EHMs), which includes palpable purpura from mixed cryoglobulinemia (MC) and porphyria cutanea tarda (PCT), but patients with advanced liver disease and cirrhosis can also manifest cutaneous stigmata of chronic liver disease:

- Easy bruising, petechiae, purpura
- Color changes of nails (leukonychia)
- Palmar erythema
- Spider angioma
- Decreased body hair (axillary and pubic hair loss)
- Generalized pruritus

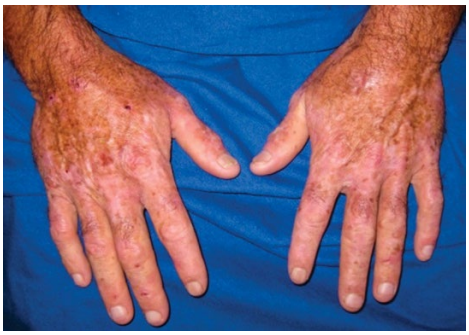


DERMATOLOGIC CONDITIONS: DERMATOLOGIC MANIFESTATIONS OF SYSTEMIC DISEASE

MC, a systemic vasculitis, is the most common dermatologic EHM. The most common symptoms of MC are weakness, arthralgias, and purpura. The dermatologic manifestations of MC vary from simple cutaneous palpable purpura to complex confluent lesions, including ulcerations of the skin.



PCT is the most common type of porphyria, frequently found in patients with have a history of chronic liver disease either due to chronic iron overload states, heavy alcohol use, or HCV. The dermatologic manifestations of PCT varies from large blisters, small vesicles, and/or milia on the dorsal aspect of the hands. Other manifestations include increased skin fragility, hypertrichosis, chronic hyper- or hypopigmentation, chloracne, sclerodermoid changes, dystrophic calcifications with ulcerations, scarring, alopecia, and onycholysis.



Management

Comanage patients with Hepatitis Central Team. Refer to Hepatitis C Care Guide.[48]

MALIGNANCY

Characteristics

Acanthosis nigricans type I: Occasionally, acanthosis nigricans is a marker of an underlying adenocarcinoma, especially of the gastrointestinal tract (60% gastric). Malignant acanthosis nigricans has a sudden onset and more extensive distribution, including the face, palms, and trunk.[17]

Cutaneous paraneoplastic syndromes. Paraneoplastic pemphigus, characterized by intractable stomatitis and blisters on the trunk and extremities, has features of pemphigus and erythema multiforme. Paraneoplastic pemphigus has a strong association with non-Hodgkin's lymphoma, chronic lymphocytic leukemia, and Castleman's disease with or without myasthenia gravis. Severe pulmonary disease (bronchiolitis obliterans) with respiratory failure is often the cause of death in affected patients.

Cutaneous metastases are uncommon, and the reported prevalence varies from 0.7% to 10% of all patients with cancer. Any solid malignancy can metastasize to the skin. Cutaneous metastases from cancers of the lung, large intestine, and kidney are most commonly found in men, whereas cancers of the breast and large intestine are the most likely primary tumors to metastasize to the skin in women. Metastases to the skin are usually flesh-colored to violaceous nodules that appear in close proximity to the primary neoplasm. The most common sites of cutaneous metastases are the scalp, neck, and trunk.

Paget's disease of the breast is an uncommon condition characterized by unilateral eczematous plaque of the nipple and areola. The disease is strongly associated with an underlying invasive carcinoma of the affected breast or ductal carcinoma in situ (DCIS). Extramammary Paget's disease is typically a persistent, eczematous plaque of the anogenital or axillary regions whose morphology and histology strongly resemble Paget's disease of the breast. Extramammary Paget's disease affects older adults and is often associated with an underlying adnexal carcinoma or an underlying cancer of the genitourinary tract or distal gastrointestinal tract.

DERMATOLOGIC CONDITIONS: DERMATOLOGIC MANIFESTATIONS OF SYSTEMIC DISEASE

Dermatological structures can also be altered as a consequence of anticancer therapies, including systemic agents, therapeutic transplants, radiotherapy (RT) and surgery. The systemic therapies most frequently associated with dermatological adverse events include cytotoxic chemotherapies, immunotherapies, biologics, targeted therapies and endocrine agents. Although most dermatological adverse events are classified grade 1 and 2 in severity, their chronicity, presence on cosmetically sensitive areas, and association with symptoms of pruritus and pain result in a need for preventive or reactive therapies.

Management

Comanage patients with the appropriate specialists, such as hematology, oncology, radiation oncology, and surgery.[49]

TREATMENT

PREVENTIVE DERMATOLOGIC CARE

Many dermatologic conditions and acute flares of chronic dermatologic diseases can be prevented or mitigated by modifiable lifestyle factors such as daily skin care, diet, sleep, exercise, stress, substance use, and smoking.

Patients should avoid frequent washing with hot water, skin irritants or triggers, and excessive sun exposure. Skin care measures include the use of alcohol-free skin moisturizers at least twice daily and the use of sun protection products, SPF ≥15 reapplied to exposed areas of body every 2 hours when outside.[25]

TREATING PRURITUS

Pruritus is a common symptom that can be caused by dermatologic and systemic conditions. Primary lesions indicate diseased skin, and secondary lesions are reactive from skin manipulation, such as excoriations and lichenification. Xerosis, or dry, scaly skin, occurs because of excessive washing of the skin or during winter during relatively low humidity.

The absence of obvious triggers or primary lesions on cutaneous examination findings can make identifying the underlying etiology of pruritus challenging. Chronic conditions, including renal and hepatic failure, thyroid disease, diabetes mellitus, pregnancy, and malignancy, can precipitate generalized pruritus without concurrent dermatologic disease. If the evaluation for multiple etiologies of pruritus is ambiguous, consider systemic, neurogenic, or psychogenic etiologies and consult with an appropriate specialist.

Inadequately treated pruritus adversely affects patient mood, quality of life, and quality of sleep. General management includes trigger avoidance, liberal emollient use, limiting water exposure, and administration of topical therapies and oral antihistamines. Targeted treatment as clinically indicated for the underlying cause of pruritus can improve symptoms.[25,27]

The sensation of pruritus is subject to intra- and inter-individual variations related to several factors such as tiredness, anxiety and stress. The intensity of itch is usually assessed by scales such as the visual scale or the numeric rating scale. The presentation of pruritus is often associated with papulopustular rash or other types of skin alterations, so the treatment of the concomitant rash can decrease the pruritic symptoms.

According to the International Forum on the Study of Itch (IFSI), chronic pruritus is defined as pruritus lasting ≥6 weeks. Chronic pruritus, often associated with other types of skin manifestations (erythema, eruption of papules and pustules, acneiform rash), can be accompanied by behavioral/adjustment disorder and a withdrawal from social life and work. [25]

Recommend the following skin care regimen for patients with pruritus:

Name	Administration
Aveeno® Moisturizing Body Wash (nonformulary)	Daily showering using mild cleanser.
Hibiclens® chlorhexidine	Rinse area with water, then apply minimum amount necessary to cover skin or wound area and wash gently. Rinse thoroughly. *Indications: wound care and general skin cleansing for affected skin of superficial and superficial partial thickness burns, hidradenitis suppurativa, procedural care, and surgical sites, as comanaged by specialists*
Thera Derm™ moisturizing lotion (canteen) or Lubrisoft moisturizing lotion (canteen) or **Eucerin® Cream Eczema Relief colloidal oatmeal**	Apply to clean, dry skin at least twice a day and within 3 minutes of washing.
Calamine lotion	Apply to clean, dry skin; avoid contact with eyes and mucous membranes; do not use on open wounds or burns. *Indications: localized areas affected by poison ivy, poison oak, poison sumac, sunburn, insect bites, or minor skin irritations*
Banana Boat Ultra Sport Sunscreen SPF 50+ Lotion (canteen) or Coppertone Sport SPF 50 Lotion (canteen)	Apply 15-30 minutes before sun exposure, then reapply at least every 40-120 minutes, as necessary

Bold = Formulary

Treatment, cont'd

If general management skin care regimen inadequately improves pruritus, consider the following treatment options:

Class	Medication	Administration
Antihistamine	Loratadine 10 mg (canteen)	Take 1 tab orally daily, as needed for urticaria, allergic contact dermatitis, and atopic dermatitis
Antihistamine	Cetirizine 10 mg (canteen)	Take 1 tab orally daily, as needed for urticaria, allergic contact dermatitis, and atopic dermatitis
Topical analgesic	**Capsaicin 0.025%**	Apply thin film to affected areas 3 to 4 times daily, as needed for chronic kidney disease (CKD)-associated pruritus
Antidepressant	**Mirtazapine 7.5-15 mg**	Take 1 tab orally nightly, as needed for MHDS patients, CKD-associated pruritus, and nocturnal pruritus
Antidepressant	**Fluvoxamine 25-150 mg**	Take 1 tab orally daily, as needed for MHDS patients, CKD-associated pruritus, and paraneoplastic pruritus
Antidepressant	**Paroxetine 10-40 mg**	Take 1 tab orally daily, as needed for MHDS patients, CKD-associated pruritus, and paraneoplastic pruritus
Antidepressant	**Sertraline 75-100 mg**	Take 1 tab orally daily, as needed for MHDS patients, CKD-associated pruritus, and cholestatic pruritus
μ-opioid receptor antagonists	**Naltrexone** 12.5-**50 mg**	Take 1 tab orally daily, as needed for patients with CKD-associated pruritus and cholestatic pruritus
Bile acid sequestrant	**Cholestyramine 4 g**	Take 1 packet orally daily, as needed for patients with cholestatic pruritus
Antineoplastic agent	Thalidomide 100 mg	Take 1 tab orally nightly, as needed for prurigo nodularis

Bold = Formulary

TOPICAL THERAPY

Topical corticosteroids, an essential therapy for inflammatory skin conditions, are classified by potency. Potency is determined by the medication, the concentration, and the type of formulation, which is available as ointments, creams, lotions, gels, foams, oils, solutions, and shampoos. The risk of adverse effects, such as atrophy, striae, rosacea, telangiectasias, purpura, and other cutaneous and systemic reactions, increases with prolonged use, a large area of application, higher potency, occlusion, and application to areas of thinner skin such as the face and genitals. Topical corticosteroids are not indicated for pruritus that is not associated with cutaneous inflammation. [25]

Topical Corticosteroids	Potency Category
Hydrocortisone 1% cream	Lowest Potency (Class 7)
Hydrocortisone 2.5% cream	Lowest Potency (Class 7)
Fluocinolone 0.01% solution	Low Potency (Class 6)
Triamcinolone 0.025% cream	Low Potency (Class 6)
Triamcinolone 0.025% ointment	Low Potency (Class 5)
Triamcinolone 0.1% cream	Moderate Potency (Class 4) *Avoid using greater than 12 weeks.*
Fluocinonide 0.05% cream, ointment, or solution	High Potency (Class 2) *Avoid using greater than 12 weeks.*
Clobetasol 0.05% cream, gel, or ointment	Very High Potency (Class 1) *Do not apply to face, axillae, or groin.* *Avoid using greater than 2 weeks.*

Bold = Formulary

Treatment, cont'd

Topical anti-infective therapy is well-tolerated. Like other topical therapies, adverse effects can include irritation, dryness and hypersensitivity reactions. Topical anti-infectives are available in formulations such as creams, ointments, lotions, and shampoos. The indications are broad, including acne vulgaris, cutaneous fungal infections, as well as skin and soft tissue infections.
Please refer to the Skin and Soft Tissue Infection Care Guide and the Chronic Wound Management Care Guide.

Topical Anti-Infectives	Type of Anti-Infective
Bacitracin/polymyxin	Antibacterial
Mupirocin 2% ointment	Antibacterial
Clotrimazole 1% cream (canteen) or **Clotrimazole 1% cream**	Antifungal
Ketoconazole 2% cream or shampoo	Antifungal
Miconazole 2% cream	Antifungal
Nystatin 100,000 units/gram cream	Antifungal
Nystatin/triamcinolone cream or ointment	Antifungal
Tolnaftate 1% cream	Antifungal
Permethrin 1% lotion	Scabicides and pediculocides
Permethrin 5% cream	Scabicides and pediculocides

Bold = Formulary

PROCEDURES DONE AT INSTITUTIONS

Procedure	Indication
Skin biopsy • Shave biopsy • Punch biopsy	• Aid in diagnosis with atypical clinical presentations • Concern for malignancy • Staging and prognosis for malignancy
Excision	• Nonmalignant lesions, such as cyst and lipoma
Wood's lamp examination	• Pigmentary disorders, such as vitiligo and melasma • Infections, such as dermatophyte infections, tinea versicolor, and erythrasma • Porphyria cutanea tarda (PCT)
Cryotherapy	• Cutaneous warts • Keloids • Seborrheic keratosis • Actinic keratosis
Intralesional corticosteroid injection with triamcinolone dilution	• Large or resistant inflammatory skin lesions, such as psoriasis plaque, inflamed cyst, acne papules, or nodules • Prurigo nodularis • Proliferative lesions, such as granulomatous wound reaction • Note: Hypertrophic or fibrotic lesions, such as hypertrophic scars and keloids, do not meet medically necessary Title 15 • Note: Alopecia areata does not meet medically necessary Title 15

Treatment, cont'd

INDICATIONS FOR REFERRAL TO DERMATOLOGY

Primary care providers can diagnose and initiate treatment for many common dermatologic conditions. Referrals to dermatology must meet medically necessary Title 15 and not for cosmetic reasons.

Diagnosis	Indications for Referral
Eczema *Atopic dermatitis* *Irritant contact dermatitis* *Allergic contact dermatitis* *Nummular eczema* *Dyshidrotic eczema* *Lichen simplex chronicus* *Seborrheic dermatitis* *Xerotic dermatitis* *Stasis dermatitis*	Initiate therapy for mild to moderate eczematous dermatoses Consider eConsult to dermatology • Uncertain diagnosis • Guidance for initiating therapy or adverse effects with therapy • Lack of response to initial therapy • Consider use of systemic agents, including biologic therapy Consider referral to dermatology • Severe, widespread, or recalcitrant disease • Lack of response to various therapies after 4 weeks Consider eConsult to allergy and immunology • Allergy testing considered
Acne vulgaris	Initiate therapy for mild to moderate acne Consider eConsult to dermatology • Uncertain diagnosis • Guidance for initiating therapy or adverse effects with therapy • Lack of response to initial therapy Consider referral to dermatology • Severe acne nodules or fulminant acne vulgaris • Acne in setting of suspected androgen excess • Isotretinoin considered • Lack of response to various therapies after 12 weeks
Acne rosacea	Initiate therapy for mild to moderate rosacea Consider eConsult to dermatology • Uncertain diagnosis • Guidance for initiating therapy or adverse effects with therapy • Lack of response to initial therapy Consider referral to dermatology • Severe rosacea • Lack of response to various therapies after 4 weeks Consider referral to ophthalmology • Ocular symptoms
Psoriasis	Initiate therapy for plaque psoriasis involving <10% total body surface area Consider eConsult to dermatology • Uncertain diagnosis • Guidance for initiating therapy or adverse effects with therapy • Lack of response to initial therapy Consider referral to dermatology • Plaque psoriasis involving ≥10% total body surface area • Pustular lesions present • Lack of response to various topical therapies after 4 weeks • Known or suspected psoriatic arthritis

Treatment, cont'd

Diagnosis	Indications for Referral
Alopecia	Consider referral to dermatology • Evidence of cicatricial alopecia
Fungal infections	Initiate appropriate antifungal therapy • Consider fungal culture for extensive involvement, tinea capitis, and symptomatic onychomycosis Consider eConsult to dermatology • Guidance for initiating systemic therapy for tinea capitis, severe tinea corporis, or onychomycosis Consider referral to dermatology • Lack of response to antifungal therapy after 4 weeks
Cutaneous lesion	Monitor benign neoplasms for changes Consider eConsult to dermatology • Expert opinion of presumably benign lesion Consider referral to dermatology • Lesion that exhibits asymmetry, irregular border, various colors, diameter larger than 6 mm, or evolving over time • Lesion that ulcerates or bleeds • Symptomatic lesion due to inflammation, pruritus, pain, irritation • More than 50 melanocytic nevi • Family history of melanoma
Concern for malignancy	Consider referral to dermatology • Biopsy or excision with margins or surgery
Patients with skin cancer or a history of skin cancer	Consider referral to dermatology
Bullous disease	Consider referral to dermatology

COMORBID MENTAL HEALTH ISSUES

The discomfort and potential for disfigurement that occurs with dermatologic conditions can contribute to low self-worth, embarrassment, and social isolation, which in turn predisposes patients to mental health issues such as anxiety, depression, and suicidal ideation. The impact of dermatologic disease on mental health is a central concern, so the timely identification and treatment of mental health issues are essential for comprehensive management.

- Consider screening for mental health conditions, such as depression and anxiety
- Recommend care coordination and collaboration with mental health as clinically indicated
- Cutaneous adverse drug reactions (CADR) occur in 2-5% of patients taking psychotropic drugs. There are also some possible drug-drug interactions between antidepressants and antipsychotics with medications used to treat skin conditions. If concerns exist, consider consultation with psychiatry.

PATIENT EDUCATION

Patient education empowers patients to make informed decisions about their treatment goals and to manage their dermatologic condition(s) by understanding:

- Risk factors/triggers and how to modify them.
- Importance of healthy lifestyle habits, including diet, exercise, and stress management.
- Medications, their purpose, potential side effects, and proper adherence.
- Diagnostic tests and how these test guide management.
- Procedures, including the benefits, risks, and alternatives to recommended procedures.

Shared decision-making is crucial, and patients should be actively involved in treatment decisions based on their individual needs, preferences, and understanding of the benefits, risks, and alternatives of various treatment options. See PE-1 and PE-2 for details.

MEDICATION TABLES

ANTIHISTAMINES

MEDICATION	DOSING*	ADVERSE EFFECTS* DRUG INTERACTIONS	COMMENTS*
Cetirizine **Zyrtec®** **Tablet: 10 mg** **$**	**Indications:** Chronic spontaneous urticaria **Dosage:** 10 mg orally once daily May increase in increments of 10 mg/day every 1-4 weeks up to 20 mg twice daily. Periodically reevaluate necessity for continued treatment	Drowsiness, headache, pharyngitis, abdominal pain, fatigue, xerostomia **Drug interactions:** Opioids, CNS depressants	**Contraindications:** Hypersensitivity to any component of formulation, levocetirizine, or hydroxyzine **Renal impairment:** CrCl >31 mL/min: No dosage adjustment needed CrCl ≤31 mL/min: 5 mg once daily **Hepatic impairment:** 5 mg once daily
Hydroxyzine pamoate **Vistaril®** **Capsule: 25 mg, 50 mg** **$**	**Indications:** Pruritus due to allergic conditions such as chronic urticaria and atopic and contact dermatoses, and in histamine-mediated pruritus **Dosage:** 25 mg orally 3-4 times daily as needed	Dry mouth, drowsiness, QT prolongation, Torsade de Pointes, urinary retention, tremor, headache, hallucination **Drug interactions:** Dronedarone, ketoconazole, MAO inhibitors, phenelzine, pimozide, thioridazine, tranylcypromine, cisapride, opioids, CNS depressants	**Contraindications:** Hypersensitivity to any component of formulation, cetirizine, or levocetirizine; prolonged QT interval; early pregnancy **Renal impairment:** CrCl >50 mL/min: No dosage adjustment needed CrCl ≤50 mL/min: A 50% dosage reduction is recommended **Hepatic impairment:** Dosage reduction may be necessary based on clinical response and degree of hepatic impairment
Loratadine **Claritin®** **Tablet: 10 mg** **$**	**Indications:** Chronic spontaneous urticaria **Dosage:** 10mg orally once daily	Headache, drowsiness, fatigue, xerostomia, stomatitis, pharyngitis **Drug interactions:** Amiodarone, haloperidol, ketoconazole, carbamazepine, cimetidine, clarithromycin, itraconazole	**Contraindications:** Hypersensitivity to any component of formulation or desloratadine **Renal impairment:** CrCl ≥30 mL/min: No dosage adjustment necessary CrCl <30 mL/minute: 10 mg every other day **Hepatic impairment:** Reduce initial dosage to 10 mg every other day

Bold = Formulary *See prescribing information for complete description of dosing, adverse effects, drug interactions, precautions, and contraindications.
The cost scale $-$$$$$ represents the relative cost of acquisition of medication only. Frequency and complexity of medication administration (institution workload, effect on adherence) should be considered when determining overall cost-effectiveness of treatment.

Medications, Cont'd

ANTIBACTERIALS			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Bacitracin/ Polymyxin Polysporin® **Topical ointment: 15 g** **$**	**Indications:** Prevention of skin and skin structure infections **Dosage:** Apply as a thin film to the affected area 1 to 3 times daily. Continue for full course of treatment; therapy should be limited to 7 days.	Rash, pruritic, erythema, edema	**Contraindications:** Hypersensitivity to any component of formulation
Mupirocin Bactroban® **Topical ointment: 2% (22 g)** **$**	**Indications:** Impetigo due to susceptible isolates of *Staphylococcus aureus (S. aureus)* and *Streptococcus pyogenes (S. pyogenes)* **Dosage:** Apply a small amount to the affected area 3 times daily for 10 days Re-evaluate patients not showing a clinical response within 3 to 5 days.	Burning, stinging or pain, itching Severe Allergic Reactions: Anaphylaxis, urticaria, angioedema, and generalized rash have been reported in patients treated with formulations of mupirocin, including mupirocin ointment	**Contraindications:** Hypersensitivity to any component of formulation Use beyond 10 days is discouraged because of the possible development of resistance. If ordered as nurse administered (NA), consider this a single-use tube; portions unused after a single dressing change per patient must be discarded.
Ceftriaxone Rocephin® **Injectable: 250 mg, 1 g** **$**	**Indications:** Skin and soft tissue infections, as well as other infections **Dosage:** 1 to 2 grams given once a day (or in equally divided doses twice a day) depending on the type and severity of infection. The total daily dose should not exceed 4 grams. Ceftriaxone therapy should be continued for at least 2 days after the signs and symptoms of infection have disappeared. The usual duration of therapy is 4 to 14 days; in complicated infections, longer therapy may be required.	Injection site induration, warmth, tightness; diarrhea, eosinophilia, thrombocytosis, Erythema multiforme, Stevens-Johnson syndrome, Toxic epidermal necrolysis **Drug interactions:** Warfarin, cyclosporine, oral contraceptives, loop diuretics, probenecid	**Contraindications:** Hypersensitivity to any component of formulation or other cephalosporins **Renal impairment:** No dosage adjustment needed **Hepatic impairment:** No dosage adjustment needed If concurrent renal and hepatic dysfunction, a reduced maximum daily dose should be considered; a maximum daily dose ≤2,000 mg/day is suggested

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Medications, Cont'd

ANTIBACTERIALS			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Azithromycin **Tablet: 250 mg, 500 mg** **$**	**Indications:** Uncomplicated skin and skin structure infections, as well as other infections **Dosage:** 500 mg orally daily for 1 day, then 250 mg orally daily for 4 days	Diarrhea, nausea, abdominal pain, vomiting **Drug interactions:** Warfarin, nelfinavir, cisapride, dronedarone, ketoconazole, pimozide, thioridazine, amiodarone	**Contraindications:** Hypersensitivity to any component of formulation, erythromycin, any macrolide, or ketolide antibiotic; history of cholestatic jaundice/hepatic dysfunction associated with prior use of azithromycin **Renal impairment:** No dosage adjustment necessary **Hepatic impairment: N**o dosage adjustment provided in the manufacturer's labeling. Use with caution due to potential for hepatotoxicity (rare)
Doxycycline **Vibramycin®** **Vibra-Tabs®** **Capsule/Tablet: 100 mg** **$**	**Indications:** Acne vulgaris, as well as infections **Dosage:** 100 mg orally every 12 hours on day 1, then 100 mg orally once daily. Continue 100 mg orally every 12 hours for severe infections	Photosensitivity, diarrhea, nasopharyngitis, nausea, dyspepsia **Drug interactions:** Warfarin, penicillin, antacids, bismuth subsalicylate, barbiturates, carbamazepine, phenytoin, methoxyflurane, oral contraceptives	**Contraindications:** Hypersensitivity to any of the tetracyclines **Renal impairment:** No dosage adjustment needed **Hepatic impairment:** No dosage adjustment needed
Tetracycline **Sumycin®** **Capsule: 250 mg, 500 mg** **$$**	**Indications:** Acne vulgaris, as well as infections **Dosage:** 1 g/day orally in divided doses, then decrease slowly to 125 mg to 500 mg orally daily or every other day	Tooth discoloration, photosensitivity, anorexia, nausea, epigastric distress, vomiting, diarrhea, erythematous rash, maculopapular rash, hepatotoxicity **Drug interactions:** Penicillin, warfarin, antacids, oral contraceptives, methoxyflurane	**Contraindications:** Hypersensitivity to any of the tetracyclines **Renal impairment:** CrCl > 90 mL/min: No dosage adjustment needed CrCl 51-90 mL/min: Administer every 8-12 hours CrCl 10-50 mL/min: Administer every 12-24 hours CrCl < 10 mL/min: Administer every 24 hours **Hepatic impairment:** No dosage adjustments provided in the manufacturer's labeling

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The cost scale $-$$$$$ represents the relative cost of acquisition of medication only. Frequency and complexity of medication administration (institution workload, effect on adherence) should be considered when determining overall cost-effectiveness of treatment

Medications, Cont'd

ANTIBACTERIALS			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Dapsone **Tablet: 100 mg** **$$-$$$**	**Indications:** Dermatitis herpetiformis **Dosage:** Initially, 50 mg orally once daily; maintenance 50 to 300 mg orally once a day. Reduce to minimum maintenance dose as soon as symptom improvement occurs	Nausea, vomiting, abdominal pains, pancreatitis, vertigo, blurred vision, tinnitus, insomnia, fever, headache, psychosis, phototoxicity, pulmonary eosinophilia, tachycardia, albuminuria, the nephrotic syndrome, hypoalbuminemia without proteinuria, renal papillary necrosis, male infertility, drug-induced Lupus erythematosus, infectious mononucleosis-like syndrome, agranulocytosis, aplastic anemia, blood dyscrasias **Drug interactions:** Rifampin, rifapentine, fluorouracil, methotrexate, pyrimethamine, saquinavir, trimethoprim	**Contraindications:** Hypersensitivity to Dapsone and/or its derivatives **Renal impairment:** No dosage adjustment needed **Hepatic impairment:** No dosage adjustments provided in the manufacturer's labeling FDA Dermatology Advisory Committee recommended that, when feasible, complete blood counts should be done weekly for the first month, monthly for six months and semi-annually thereafter

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Medications, Cont'd

ANTIFUNGALS			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Clotrimazole Lotrimin® **Topical cream: 1%** **$**	**Indications:** Cutaneous candidiasis, tinea corporis, tinea cruris, tinea pedis, tinea versicolor **Dosage:** Apply to affected and surrounding area(s) twice daily	Blistering, burning, edema, erythema, generalized skin irritation, peeling, pruritus, stinging, urticaria	**Contraindications:** Hypersensitivity to any component of formulation
Ketoconazole Nizoral® **Topical cream, shampoo: 2%** **$**	**Indications:** Cutaneous candidiasis, tinea corporis, tinea cruris, tinea pedis, tinea versicolor, seborrheic dermatitis **Dosage:** Cutaneous candidiasis, tinea corporis, tinea cruris, tinea versicolor (cream): Apply to affected and surrounding area(s) once daily for 2 weeks Tinea pedis (cream): Apply to affected and surrounding area(s) once daily for 6 weeks Seborrheic dermatitis (cream): Apply to the affected area twice daily for 4 weeks or until clinical clearing Tinea versicolor (shampoo): Apply to damp skin and surrounding area(s), lather, leave on skin for 5 minutes then rinse	Local skin irritation, pruritus, stinging	**Contraindications:** Hypersensitivity to any component of formulation
Miconazole Micatin® **Topical cream: 2% (30 g)** **$**	**Indications:** Cutaneous candidiasis, tinea pedis, tinea cruris, tinea corporis **Dosage:** Cutaneous candidiasis, tinea cruris: Apply a thin layer topically to the affected skin area(s) twice daily for 2 weeks Tinea pedis, tinea corporis: Apply a thin layer topically to the affected skin area(s) twice daily for 4 weeks	Contact dermatitis	**Contraindications:** Hypersensitivity to any component of formulation
Nystatin Mycostatin® **Topical cream: 100,000 units/g (30g)** **$**	**Indications:** Cutaneous and mucocutaneous candidiasis **Dosage:** Apply liberally to the affected skin area(s) twice daily until healing is complete	Allergic reactions, burning, itching, rash, eczema, pain on application	**Contraindications:** Hypersensitivity to any component of formulation

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Medications, Cont'd

ANTIFUNGALS			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Nystatin/ triamcinolone Mycolog II® **Topical cream, ointment: 30g** **$**	**Indications:** Cutaneous candidiasis **Dosage:** Apply sparingly as a thin film to the affected skin twice daily	Burning, itching, skin irritation, dryness, folliculitis, hypertrichosis, acneiform eruptions, hypopigmentation, allergic contact dermatitis	**Contraindications:** Hypersensitivity to any component of formulation
Tolnaftate Tinactin® **Topical cream: 1%** **$**	**Indications:** Tinea pedis, tinea corporis, tinea cruris **Dosage:** Tinea pedis, tinea corporis, tinea cruris (treatment): Apply a thin layer over affected area twice daily Tinea pedis (prevention): Apply once or twice daily	Local skin irritation, contact dermatitis, pruritus, stinging of the skin	**Contraindications:** Hypersensitivity to any component of formulation
Fluconazole Diflucan® **Tablet: 100 mg, 150 mg, 200 mg** **$-$$**	**Indications (off-label):** Tinea barbae, tinea infection (extensive or refractory to topical therapy) **Dosage:** Tinea corporis/tinea cruris: 150-200 mg orally once weekly for 2 to 4 weeks Tinea pedis/tinea manuum: 150 mg orally once weekly for 2 to 6 weeks Tinea versicolor (pityriasis versicolor): 300 mg orally once weekly for 2 weeks Tinea barbae: 200 mg orally once daily for 4 to 6 weeks	Nausea, vomiting, headache, prolonged QT interval, enterocolitis, abdominal pain, elevated hepatic enzymes, diarrhea, rash **Drug interactions:** Amiodarone, amitriptyline, nortriptyline, quinidine, carbamazepine, celecoxib, warfarin, cyclosporin, clozapine, atorvastatin, simvastatin, lurasidone, tacrolimus, erythromycin, voriconazole, alfuzosin, pimozide	**Contraindications:** Hypersensitivity to any component of formulation; Coadministration of other drugs known to prolong the QT interval and which are metabolized via the enzyme CYP3A4 (e.g., erythromycin, pimozide, quinidine, etc.) **Renal impairment:** CrCl > 50 mL/min: No dosage adjustment needed CrCl ≤ 50 mL/min: Administer 50% of usual dosage **Hepatic impairment:** No dosage adjustment needed; use with caution

Bold = Formulary *See prescribing information for complete description of dosing, adverse effects, drug interactions, precautions, and contraindications. The cost scale $-$$$$$ represents the relative cost of acquisition of medication only. Frequency and complexity of medication administration (institution workload, effect on adherence) should be considered when determining overall cost-effectiveness of treatment.

Medications, Cont'd

ANTIVIRALS			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Acyclovir Zovirax® **Tablet: 400 mg, 800 mg** **$**	**Indications:** Herpes zoster, varicella **Dosage:** Herpes zoster: 800 mg orally every 4 hours, 5 times daily for 7 to 10 days Varicella: 800 mg orally 4 times daily for 5 days	Nausea, vomiting, malaise, diarrhea, headache **Drug interactions:** Probenecid, cidofovir, foscarnet, tizanidine, fezolinetant, varicella virus vaccine, live/attenuated zoster vaccine	**Contraindications:** Hypersensitivity to acyclovir or valacyclovir **Renal impairment:** CrCl >25 mL/min/1.73 m^2: No dosage adjustment needed CrCl 10-25 mL/min/1.73 m^2: 800 mg orally every 8 hours CrCl <10 mL/min/1.73 m^2: 800 mg orally every 12 hours **Hepatic impairment:** No dosage adjustment needed

SCABICIDES & PEDICULOCIDES			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Permethrin Nix®, Elimite® **Topical cream: 5% (60 g)** **Topical lotion: 1% (2 oz)** **$**	**Indications:** Scabies, pediculosis capitis **Dosage:** Scabies (cream): Apply to all areas of the body from the neck to soles of feet (30 g for average adult); leave on for 8 to 14 hours before removing by washing (shower or bath). For older adults, also apply on the hairline, neck, scalp, temple, and forehead. One application is generally curative; may repeat if living mites are observed 14 days after first treatment Pediculosis capitis (lotion): Apply to damp hair that has just been shampooed with a non-conditioning shampoo; saturate hair and scalp beginning behind the ears and at back of neck; leave on 10 minutes; rinse with warm water; remove nits with nit comb; repeat application if live lice present 7 days after initial treatment	Burning, stinging, pruritic, erythema, numbness, tingling, rash	**Contraindications:** Hypersensitivity to any of its components, to any synthetic pyrethroid or pyrethrin

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Medications, Cont'd

LOCAL ANTI-INFECTIVES			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Chlorhexidine Hibiclens® **Topical cleanser: 4% (120 mL)** **$**	**Indications:** Skin wound and general skin cleansing **Dosage:** Apply minimum amount necessary to cover the affected skin or wound area and wash gently, then rinse thoroughly	Allergic sensitization, erythema, hypersensitivity reaction, rough skin, xeroderma	**Contraindications:** Hypersensitivity to any component of formulation
Selenium sulfide Selsun® **Topical lotion: 2.5%** **$**	**Indications:** Tinea versicolor, seborrheic dermatitis of the scalp, dandruff **Dosage:** Tinea versicolor: Apply to affected areas and lather with a small amount of water. Allow product to remain on skin for 10 minutes, then rinse the body thoroughly. Repeat this procedure once a day for seven days Seborrheic dermatitis, dandruff: Apply to wet scalp and massage in. Leave on for 2 to 3 minutes. Rinse thoroughly. Two applications per week for 2 weeks usually brings control. After 2 weeks, the shampoo may be used less frequently as needed	Skin irritation, hair loss, discoloration of hair, oiliness or dryness of hair and scalp	**Contraindications:** Hypersensitivity to any component of formulation

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Medications, Cont'd

ANTI-INFLAMMATORY AGENTS			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Clobetasol Temovate® **Topical cream, gel, ointment: 0.05% (15 g, 30 g)** **Topical solution: 0.05% (25 mL)** **$**	**Indications:** Corticosteroid-responsive dermatoses **Dosage:** Apply a thin layer topically to the affected skin area(s) 2 times daily for up to 2 weeks. Max: 50 g/week or 50 mL/week	Burning, stinging, itching, skin atrophy, erythema, folliculitis, xeroderma, cracking and fissuring of the skin, dryness, hypertrichosis, acneiform eruptions, hypopigmentation, perioral dermatitis, allergic contact dermatitis, secondary infection, skin irritation, striae, miliaria	**Contraindications:** Hypersensitivity to any component of formulation Very High Potency
Fluocinolone Synalar® **Topical solution: 0.01% (60 mL)** **$**	**Indications:** Corticosteroid-responsive dermatoses **Dosage:** Apply a thin layer topically to the affected skin area(s) 2 to 4 times daily	Burning, itching, skin irritation, dryness, folliculitis, hypertrichosis, acneiform eruptions, hypopigmentation, secondary infection, skin atrophy, striae, miliaria	**Contraindications:** Hypersensitivity to any component of formulation Low Potency
Fluocinonide Lidex® **Topical cream, ointment: 0.05% (15 g, 30 g, 60 g)** **Topical solution: 0.05% (60 mL)** **$**	**Indications:** Corticosteroid-responsive dermatoses **Dosage:** Apply a thin layer topically to the affected skin area(s) 2 to 4 times daily	Burning, itching, skin irritation, dryness, folliculitis, hypertrichosis, acneiform eruptions, hypopigmentation, secondary infection, skin atrophy, striae, miliaria	**Contraindications:** Hypersensitivity to any component of formulation High Potency
Hydrocortisone Cortaid® **Topical cream: 1%, 2.5%** **$**	**Indications:** Corticosteroid-responsive dermatoses **Dosage:** Apply a thin layer topically to the affected skin area(s) 2 to 4 times daily	Burning, itching, skin irritation, dryness, folliculitis, hypertrichosis, acneiform eruptions, hypopigmentation, secondary infection, skin atrophy, striae, miliaria	**Contraindications:** Hypersensitivity to any component of formulation Low Potency
Triamcinolone Kenalog® **Topical cream: 0.025% (15 g, 80 g, 454 g), 0.1% (15 g, 30 g, 80 g)** **Topical ointment: 0.025% (15 g, 80 g)** **$**	**Indications:** Corticosteroid-responsive dermatoses **Dosage:** Cream/ointment 0.025%: Apply a thin layer topically to the affected skin area(s) 2 to 4 times daily Cream 0.1%: Apply a thin layer topically to the affected skin area(s) 2 to 3 times daily	Burning, itching, skin irritation, dryness, folliculitis, hypertrichosis, acneiform eruptions, hypopigmentation, secondary infection, skin atrophy, striae, miliaria	**Contraindications:** Hypersensitivity to any component of formulation Low to Medium Potency (depends on delivery vehicle)

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Medications, Cont'd

KERATOLYTIC AGENTS			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Benzoyl peroxide Benzac AC® **Topical gel: 10%** **$**	**Indications:** Acne vulgaris **Dosage:** Apply a thin layer topically to the affected skin area(s) 1 to 3 times daily. Reduce application to once daily or every other day if bothersome dryness or peeling occurs.	Erythema, burning, itching, dryness, peeling, edema, allergic contact dermatitis, photosensitivity **Drug interactions:** Photosensitizing agents, topical dapsone	**Contraindications:** Hypersensitivity to any component of formulation
Salicylic acid Duofilm® **Topical liquid: 17% (9 mL)** **$**	**Indications:** Common and plantar warts **Dosage:** Apply topically to sufficiently cover each wart after washing and drying affected area; procedure may be repeated once or twice daily as needed for up to 12 weeks	Local irritation, desquamation, exfoliation of skin **Drug interactions:** Calcipotriene	**Contraindications:** Hypersensitivity to any component of formulation

OTHER TOPICAL AGENTS			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Calcipotriene Dovonex® **Topical cream: 0.005%** **$$$$**	**Indications:** Plaque psoriasis **Dosage:** Apply a thin layer topically to the affected skin twice daily Max: 100 g/week/m^2	Burning, pruritus, rash, skin irritation, rash, stinging, tingling of skin **Drug interactions:** Topical hydrocortisone valerate, vitamin D analogs, cardiac glycosides, salicylic acid	**Contraindications:** Hypersensitivity to any component of formulation; hypercalcemia or vitamin D toxicity Not to be used on face
Colloidal oatmeal Eucerin® Eczema Relief Cream **Topical cream: 1% (8 oz)** **$**	**Indications:** Temporarily protects and helps relieve minor skin irritation and itching due to rashes, eczema **Dosage:** Apply topically as needed	Burning, stinging, redness, skin irritation	**Contraindications:** Hypersensitivity to any component of formulation

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Medications, Cont'd

OTHER TOPICAL AGENTS			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Fluorouracil Efudex® **Topical cream: 5%** **$$**	**Indications:** Actinic or solar keratoses, superficial basal cell carcinomas **Dosage:** Apply topically twice daily Actinic or solar keratoses: Continue treatment until the inflammatory response reaches the erosion stage, at which time use of the drug should be terminated. Usual duration of therapy is 2 to 4 weeks Superficial basal cell carcinomas: Continue treatment for at least 3 to 6 weeks. Therapy may be required for as long as 10 to 12 weeks before the lesions are obliterated	Burning, crusting, allergic contact dermatitis, pruritus, scarring, rash, soreness, ulceration, leukocytosis, miscarriage **Drug interactions:** Aminolevulinic acid, brivudine, sorivudine	**Contraindications:** Hypersensitivity to any component of formulation; dihydropyridine dehydrogenase (DPD) enzyme deficiency; pregnancy
Imiquimod Aldara® **Topical cream: 5%** **$**	**Indications:** Actinic keratosis, superficial basal cell carcinomas **Dosage:** Actinic keratosis: Apply topically to defined treatment area 2 times per week at bedtime for 16 weeks; leave on skin for 8 hours Superficial basal cell carcinomas: Apply topically once daily 5 times per week for 6 weeks; apply at bedtime and leave on skin for 8 hours	Application site reaction, burning, edematous skin, erythema, peeling, pruritus, scabbing, skin discharge, skin ulcers, influenza-like illness, headache, upper respiratory infection	**Contraindications:** Hypersensitivity to any component of formulation
Moisturizing lotion TheraDerm™ **Topical lotion: 8 oz** **$**	**Indications:** Dry skin **Dosage:** Apply topically as often as needed	Burning, stinging, redness, skin irritation	**Contraindications:** Hypersensitivity to any component of formulation
Zinc oxide **Topical ointment: 20% (30 g, 60 g)** **$**	**Indications:** Skin protectant **Dosage:** Apply topically as often as needed	None	**Contraindications:** Hypersensitivity to any component of formulation

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Medications, Cont'd

NONFORMULARY THERAPIES			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
Coal tar Thera-Gel® Topical shampoo: 0.5%, 2% $	**Indications:** Seborrheic dermatitis; psoriasis of the scalp **Dosage:** Apply to wet hair and massage into lather at least twice weekly; leave on for several minutes, then rinse. Alternately, may apply topically to the psoriatic scalp area(s) at least twice weekly; wash off after 5 minutes, initially and gradually increase contact time up to 1 hour	Staining, skin irritation, photosensitivity, folliculitis, contact dermatitis	**P&T Approved Non-Formulary Use Criteria:** Seborrheic dermatitis; psoriasis of the scalp **Contraindications:** Hypersensitivity to any component of formulation Coal tar preparations should not be applied to patients with skin abrasion or skin that is inflamed, broken, or infected
Diphenhydramine Benadryl® Capsule: 25 mg, 50 mg $	**Indications:** Urticarial eruptions **Dosage:** 25 to 50 mg orally 3 to 4 times daily as needed Max: 300 mg/day	Drowsiness, restlessness, xerostomia, asthenia, headache, dizziness **Drug interactions:** Monoamine oxidase inhibitors, phenelzine, thioridazine, tranylcypromine, CNS depressants (hypnotics, sedatives, tranquilizers, etc.)	**P&T Approved Non-Formulary Use Criteria:** Restricted to urticarial eruptions, prophylaxis and treatment of extrapyramidal movement disorders and syndromes in psychiatric patients, and emergency use (anaphylaxis, acute agitation, etc.) **Contraindications:** Hypersensitivity to diphenhydramine hydrochloride and other antihistamines of similar chemical structure **Renal impairment:** No dosage adjustment needed **Hepatic impairment:** No dosage adjustment needed, use lowest effective dose and as needed dosing only

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Medications, Cont'd

NONFORMULARY THERAPIES			
MEDICATION	**DOSING***	**ADVERSE EFFECTS* DRUG INTERACTIONS**	**COMMENTS***
A&D ointment Topical ointment $	**Indications:** Skin protectant **Dosage:** Apply topically as often as needed	None	**P&T Approved Non-Formulary Use Criteria:** 1) Severe chronic xerosis with potential of significant sequalae, or 2) Prevention of cheilitis in patients with intermaxillary fixation **Contraindications:** Hypersensitivity to any component of formulation
Silicone scar gel Scaraway® Gel Topical gel $	**Indications:** Scars **Dosage:** Apply a thin layer topically twice daily	Rash	**P&T Approved Non-Formulary Use Criteria:** • Recommended use by a gender-affirming surgeon (i.e., post gender-affirming chest surgery) • Limited use to a maximum of 6 months **Contraindications:** Hypersensitivity to any component of formulation
Podofilox Condylox® Topical solution: 0.5% $$	**Indications:** External genital warts (solution) **Dosage:** Apply a thin layer topically to wart(s) twice daily for 3 days, followed by 4 days of no therapy. May repeat this 1-week cycle until there is no visible wart tissue or for a maximum of 4 cycles. Max: 0.5 mL/day (or ≤10 cm^2 of wart tissue) Note**:** Discontinue after 4 treatment cycles if incomplete response and consider alternative treatment; do not repeat use.	Burning, pain, inflammation, erosion, itching **Drug interactions:** Imiquimod, podophyllum	**Contraindications:** Hypersensitivity to any component of formulation
Emollient body wash Aveeno® Daily Moisturizing Body Wash Topical liquid $	**Indications**: Dry skin **Dosage:** Work into a rich, creamy lather, then rinse	Skin irritation	**Contraindications:** Hypersensitivity to any component of formulation

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Patient Education

Skin Care For Everyone

Protect Your Skin

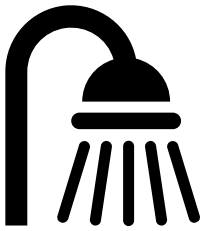
Protect yourself from the sun

- Use sunscreen. Use a palm-sized amount of broad-spectrum sunscreen with an SPF of at least 30. Put on more sunscreen every two hours or more often if you sweat.
- Seek shade. Try to stay out of the sun between 10 a.m. and 4 p.m. That's when the sun's ultraviolet (UV) rays are strongest.
- Wear protective clothing. Cover your skin with long-sleeved shirts, long pants, and a hat or cap.

Don't smoke

- Smoking narrows the tiny blood vessels in the outermost layers of skin. This lessens blood flow and depletes the skin of oxygen and nutrients that are key for skin health.
- Smoking damages fibers called collagen and elastin that give skin strength
- Smoking also raises the risk of squamous cell skin cancer, especially on the lips. If you smoke, the best way to protect your skin is to quit

Wash your face daily and after exercise



- Wash your face with a mild facial cleanser and warm — not hot — water
- Before you shave, wet your skin and hair to soften it
- Do not stretch your skin taut while shaving
- Shave in the direction of hair growth
- Rinse after each swipe of the razor
- Change your blade after five to seven shaves to minimize irritation.

Moisturize daily

Moisturizer works by trapping water in your skin to prevent it from drying out and getting itchy.

- Apply moisturizer to your face and body immediately after bathing, showering or shaving while the skin is still damp
- Use lotion on your hands after washing.

For patients with skin conditions

See your provider if you have a new skin irritation or rash so they can diagnose the problem and start treatment.

- Use the treatment recommended by your provider as directed. Most of the time, your provider will recommend a cream, ointment, or shampoo for your skin condition. Other times, your provider will recommend taking a pill or even an injection to treat certain skin conditions.
- Follow up with your provider as recommended so that different treatments can be started if one is not working for you.

Patient Education

Skin Self Exam

What You Should Know

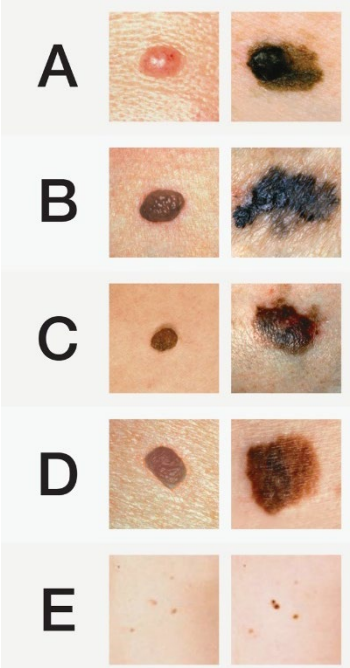
Can I get skin cancer?

- Anyone can get skin cancer, including those with brown and black skin.
- Even if you never sunburn, you can get skin cancer.
- When found early, most skin cancers can be cured.

What are the symptoms and signs of skin cancer?

Many people have no symptoms. For those who do, look for the following:

- New dark spot or darker patch of skin that is growing, bleeding, or changing in any way.
- Sore that does not heal, or sore that heals and comes back.
- Sore that has a hard time healing, especially if the sore appears in a scar or on skin that was injured in the past
- New dark line underneath or around a fingernail or toenail



Remember your ABCDEs when looking at a skin spot or skin patch

A. Asymmetry: If you draw a line in the middle, does one half not match the other half?
B. Border: Do the borders of the look uneven or irregular in shape?
C. Color: Is the skin spot or skin patch made up of more than one color?
D. Diameter: Is the size of the skin spot or skin patch larger than a pencil eraser?
E. Evolving: Is the skin spot or skin patch changing in size, shape, or color over time?

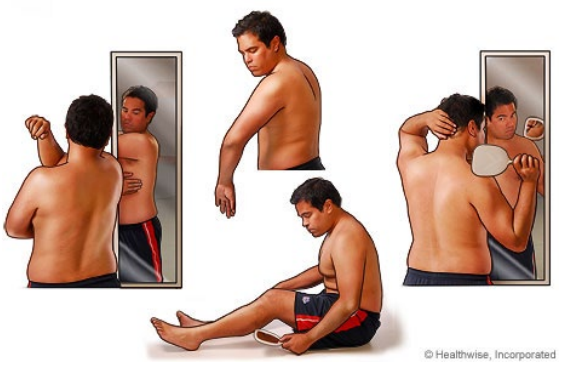
If you answered "yes" to any of the above questions, contact your health care team.

How do I do a skin self-exam?

Use a mirror to help the total body exam of your skin:

- Look from head to toe
- Check hard-to-see areas, like the top of your head and back using the mirror
- Check the bottoms of your feet, your fingernails and toenails, the inside of your mouth, your groin, and your buttocks
- Check inside your mouth

Repeat a skin self-exam every month. Contact your health care team if you have concerns for skin cancer.



How can I prevent skin cancer?

Most skin cancers are caused by too much sun exposure, so you can prevent skin cancer by doing the following:

- Seek shade whenever possible
- Wear clothes that protect your skin from the sun.
- Wear a hat or cap for shade.
- Wear broad-spectrum sunscreen that is SPF 30 or higher. Reapply to your skin every 2 hours when you're in the sun. Reapply more often if you're sweating.

Educación del Paciente

Cuidado de la piel para todos

Lo que debes saber

Protégete del sol

- Usar protector solar. Use una cantidad de protector solar de amplio espectro del tamaño de la palma de la mano con un SPF de al menos 30. Aplícate más protector solar cada dos horas o con más frecuencia si sudas.
- Busca la sombra. Trate de mantenerse alejado del sol entre las 10 a.m. y las 4 p.m. Es entonces cuando los rayos ultravioletas (UV) del sol son más fuertes.
- Use ropa protectora. Cúbrase la piel con camisas de manga larga, pantalones largos y un sombrero o gorra.

No fumes

- Fumar estrecha los pequeños vasos sanguíneos de las capas más externas de la piel. Esto disminuye el flujo sanguíneo y agota el oxígeno y los nutrientes que son clave para la salud de la piel.
- Fumar daña las fibras llamadas colágeno y elastina que le dan fuerza a la piel
- Fumar también aumenta el riesgo de cáncer de piel de células escamosas, especialmente en los labios. Si fumas, la mejor manera de proteger tu piel es dejar de hacerlo

Lávate la cara diariamente y después de hacer ejercicio

- Lávate la cara con un limpiador facial suave y agua tibia, no caliente
- Antes de afeitarse, humedezca la piel y el vello para suavizarlo
- Afeitarse en la dirección del crecimiento del vello
- Enjuague después de cada pasada de la maquinilla de afeitar
- Cambie la cuchilla después de cinco a siete afeitados para minimizar la irritación.

Hidratar diariamente

- El humectante funciona atrapando el agua en la piel para evitar que se seque y pique.
- Aplique crema hidratante en la cara y el cuerpo inmediatamente después de bañarse, ducharse o afeitarse mientras la piel aún está húmeda
- Use loción en las manos después de lavarse.

Para pacientes con afecciones de la piel

Consulte a su proveedor si tiene una nueva irritación o erupción cutánea para que pueda diagnosticar el problema y comenzar el tratamiento.

- Use el tratamiento recomendado por su proveedor según las indicaciones.
- La mayoría de las veces, su proveedor le recomendará una crema, ungüento o champú para la afección de su piel. Otras veces, su proveedor le recomendará tomar una pastilla o incluso una inyección para tratar ciertas áreas afectadas en la piel.
- Haga un seguimiento con su proveedor según lo recomendado para que se puedan iniciar diferentes tratamientos si uno no funciona para usted.

Educación del Paciente

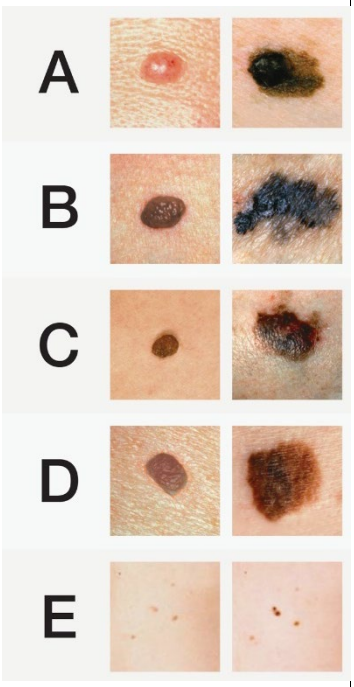
Autoexamen de la piel

Lo que debes saber

¿Puedo conseguir cáncer de la piel?

- Cualquier persona puede contraer cáncer de piel, incluidas las personas de piel morena y negra.
- Incluso si nunca se quema con el sol, puede contraer cáncer de piel.
- Cuando se detectan a tiempo, la mayoría de los cánceres de piel se pueden curar.

¿Cuales son los sintomas y signos de cancer de la piel?



Muchas personas no presentan síntomas. Para aquellos que sí lo tienen, busque lo siguiente:

- Nueva mancha oscura o parche más oscuro de la piel que está creciendo, sangrando o cambiando de alguna manera.
- Llaga que no cicatriza o llaga que sana y vuelve
- Llaga que tiene dificultades para sanar, especialmente si la llaga aparece en una cicatriz o en la piel que se lesionó en el pasado
- Nueva línea oscura debajo o alrededor de una uña de la mano o del pie

Recuerde sus ABCDE cuando vea una mancha o un parche cutáneo

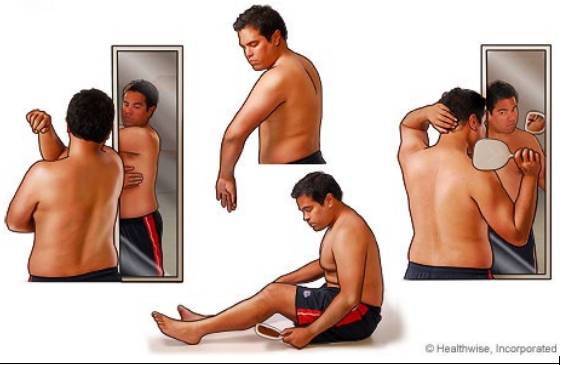
A. Asimetría: ¿Si dibujas una línea en el medio, una mitad no coincide con la otra mitad?
B. Borde: ¿Los bordes del borde se ven desiguales o irregulares en forma?
C. Color: ¿La mancha o el parche cutáneo se compone de más de un color?
D. Diámetro: ¿El tamaño de la mancha o parche cutáneo es mayor que el de la goma de borrar de un lápiz?
E. Evolución: ¿La mancha o el parche cutáneo cambia de tamaño, forma o color con el tiempo?

Como hago un autoexamen de la piel?

Use un espejo para ayudar a realizar el examen corporal completo de su piel:

- Mira de pies a cabeza
- Revise las áreas difíciles de ver, como la parte superior de la cabeza y la espalda, usando el espejo
- Revise las plantas de los pies, las unas de las manos y de los pies, el interior de la boca, la ingle y los glúteos.
- Revise el interior de la boca

Repita un autoexamen de la piel todos los meses. comunícate con tu equipo de atención medica si te preocupa el cáncer de la piel.



¿Como puedo prevenir el cáncer de la piel?

La mayoría de los canceres de la piel son causadas por la exposición excesiva al sol, por lo que puede prevenir el cáncer de la piel haciendo lo siguiente:

- Busque la sombra siempre que sea posible
- Use ropa que proteja su piel del sol.
- Use un sombrero o gorra para tener sombra.
- Use protector solar de amplio espectro con SPF 30 o superior. Vuelva a aplicar sobre la piel cada dos horas cuando estes en el sol. Aplíquelo con más frecuencia si está sudando.