Irritant Contact Dermatitis
There are two major forms of contact dermatitis: irritant and allergic.

Irritant contact dermatitis (ICD) is a cutaneous inflammatory disorder resulting from activation of the innate immune system by direct cytotoxic effect of a chemical or physical agent, whereas allergic contact dermatitis is a delayed-type hypersensitivity immune reaction mediated by hapten-specific T cells.

ICD is the most common form of occupational skin disease, estimated to constitute between 70% and 80% of all occupational skin disorders.
Clinical manifestations of ICD are determined by the properties of the irritating substance as well as host and environmental factors.

These include concentration, pH, mechanical pressure, temperature, humidity, and duration of contact.

Low ambient humidity and cold are important factors in decreasing the water content of the stratum corneum and, consequently, increasing the permeability to irritants such as soaps, detergents, acids, bases, and solvents.
Occlusion, excessive humidity, and maceration increase the water content of the stratum corneum, with consequent enhanced percutaneous absorption of water-soluble substances.

In addition, irritated skin may become more susceptible to superimposed allergic sensitization.

Important predisposing characteristics of the individual include age, sex, pre-existing skin disease, anatomic region exposed, and sebaceous activity.
There are age-associated changes in the skin that can alter the skin’s response to irritants.

Both infants and the elderly are more often affected by ICD because of their less robust epidermal barrier, and they also develop more severe symptoms.

While skin irritation may be seen more often on the upper extremities of women than men, this higher prevalence of ICD may be due to increased frequency of exposure rather than inherent gender differences.
Genetic factors also play a role.

Patients with a history of atopic dermatitis have a 13.5 times greater risk of developing occupational dermatitis.

Lastly, the most commonly affected sites are exposed areas such as the hands and the face, with hand involvement seen in ~80% of patients and facial involvement in 10%.

Excessive exposure to water, soaps and detergents, common causes of ICD, play a critical role given that wet work (immersion in water for >2 hours, occlusive protective gear, and/or hand washing >20 times/day) represents one of the most important risk factors for developing irritant dermatitis.
Although the cellular mechanisms of ICD remain elusive, increasing evidence suggests that activated keratinocytes act as signal transducers in the control of host homeostatic responses to exogenous stimuli and they serve as key immunoregulators. While other mediators such as prostaglandins, leukotrienes, and neuropeptides may possibly play a role, cytokines carry the most interest in ICD as they are the central mediators in T-cell recruitment and inflammation.
IRRITANTS AND MECHANISMS OF TOXICITY

- **Detergents**  
  Barrier disruption, protein denaturation, membrane toxicity

- **Acids**  
  Protein denaturation, cytotoxicity

- **Alcohols**  
  Protein denaturation

- **Alkalis**  
  Barrier lipid denaturation, cytotoxicity through cellular swelling

- **Oils**  
  Disorganization of barrier lipids
Organic solvents: Solubilization of membrane lipids, membrane toxicity
Oxidants: Cytotoxicity
Water: If barrier is disrupted, cytotoxicity through swelling of viable epidermal cells.
CLINICAL FEATURES

Acute Irritant Contact Dermatitis

- *Acute* ICD, commonly seen with occupational accident develops when the skin is exposed to a potent irritant.
- The acute reaction reaches its peak quickly, usually within minutes to hours after exposure, and then starts to heal.
- This is termed the decrescendo phenomenon.
Symptoms of acute ICD include burning, stinging, and soreness of the directly affected sites.

Physical signs include erythema, edema, bullae, and possibly necrosis.

These lesions are restricted to the area where the irritant or toxicant damaged the tissue, with sharply demarcated borders and asymmetry pointing to an exogenous cause.

If there is no dermal injury, healing should be complete. The potent irritants that most frequently lead to ICD are acids and alkalis, resulting in chemical burns.
Acute Delayed Irritant Contact Dermatitis

*Acute delayed* ICD is a retarded inflammatory response characteristic of certain irritants, such as anthralin.

Adverse reactions to these chemicals are considered idiosyncratic, except when they are applied to previously injured skin, e.g. sites of xerosis or atopic dermatitis.

Clinically visible inflammation is not seen until 8 to 24 hours (or more) after exposure, and thus may mimic allergic contact dermatitis; however, the associated symptom is more frequently burning rather than pruritus.
Irritant Reaction Irritant Contact Dermatitis

- *Irritant reaction* ICD is a type of subclinical irritant dermatitis in individuals exposed to wet chemical environments, such as hairdressers, those with frequent exposures to soap and water.

- It is characterized by one or more of the following signs: scaling, redness, vesicles, pustules and erosions, often beginning under occlusive jewelry (e.g. rings) and then spreading onto the fingers and then the hands and the forearms.
Asteatotic Dermatitis

- *Asteatotic dermatitis*, also referred to as astematotic eczema, eczema craquelé, is a special variant seen primarily during dry winter months.
- Elderly individuals who frequently bathe without remoisturizing are at particular risk of developing astematotic dermatitis.
- Intense pruritus is common, with the skin appearing dry
- with ichthysiform scale and characteristic patches of superficially cracked skin.
Traumatic Irritant Contact Dermatitis

Traumatic ICD may develop after acute skin trauma, such as from burns, lacerations, or acute ICD. Patients should be asked whether they have cleansed the skin with strong soaps or detergents.

It is characterized by eczematous lesions, most commonly on the hands, that last for weeks to months with persistent redness, infiltration, scale, and fissuring in the affected areas.
Pustular and Acneiform Irritant Contact Dermatitis

- *Pustular and acneiform* ICD results from exposure to certain irritants, such as metals, mineral oils, tars, greases, and naphthalenes.

- This syndrome should be considered in conditions in which folliculitis or acneiform lesions develop in settings outside of typical acne, particularly in patients with atopic dermatitis, seborrheic dermatitis, or prior acne vulgaris.

- The pustules are “sterile” and transient.
Airborne Irritant Contact Dermatitis

Airborne ICD develops in irritant-exposed sensitive skin of the face and periorbital regions. While this often simulates photoallergic reactions, involvement of the upper eyelids, philtrum, and submental regions in patients with airborne ICD may aid in distinguishing between these two entities.

Airborne ICD results from exposure to floating dusts, fibers (particularly fiberglass), and volatile solvents and sprays.
Classification of Irritant Chemicals

**Acids**

A variety of both inorganic and organic acids can be corrosive to the skin. Acids cause epidermal damage via protein denaturation and cytotoxicity. Principally, all strong acids give the same clinical features, including erythema, vesication, and necrosis.

Hydrofluoric acid and sulfuric acid cause the most severe burns, even at low concentrations, and there can be significant absorption leading to systemic toxicity.
In general, the organic acids tend to be less irritating. Among the organic acids, acetic, acrylic, formic, glycolic, benzoic, and salicylic acids are the most common irritants, particularly after prolonged exposure.
Alkalis

- Alkalis or bases often cause more painful and severe damage than most acids.
- There are generally no vesicles, but rather necrotic skin that first appears dark brown, then black, and ultimately becomes hard, dry, and cracked.
- Alkalis disrupt barrier lipids and denature proteins with subsequent fatty acid saponification, thus subjecting the cell to edema and resultant cytotoxicity.
- Strong alkalis include sodium, ammonium, calcium, and potassium hydroxide; sodium and potassium carbonate; and calcium oxide.
Alcohols/glycols

- Alcohols are used widely as solvents, disinfectants, preservatives in cosmetics, and penetration enhancers in drug delivery systems.

- Alcohols are the safest known topical antiseptic compounds, providing bactericidal activity against most Gram-positive and Gram-negative bacteria as well as many fungi and viruses.

- Most appropriate for this use are diluted solutions of ethyl alcohol, propyl alcohol, and isopropyl alcohol, which act by means of protein denaturation.
Propylene glycol can produce both allergic and irritant contact dermatitis and sources of exposure include personal care products, topical corticosteroids, and other topical medications.
**Bodily fluids**

- Urine, feces (especially in the setting of diarrhea), and saliva can lead to ICD. In babies, irritant diaper dermatitis is a common problem and is often characterized by glazed erythema of convex surfaces and at the diaper margins, with sparing of the skin folds; edema, scaling, and superficial erosions may be observed.

- Incontinence can lead to similar problems in the elderly.
Avoidance of causative irritants in the home or the workplace is the primary treatment for ICD.

Strategies in the prevention of ICD include the identification of irritants with appropriate substitution, the establishment of engineering controls to reduce exposure, the utilization of personal protective equipment such as gloves and special clothing, and barriers such as ointments, emollients, or creams.
Preventive skin care at the workplace incorporates pre-exposure protection by application of protective creams to intact skin, removal of irritants by mild cleaning agents, and enhancement of barrier function generation by emollients or moisturizers.

Non-irritating fatty substances such as petrolatum preclude hydrophilic chemical penetration and restore barrier function.
The goal of treatment is to restore normal epidermal barrier function.

Topical corticosteroids are frequently used, but their efficacy has been controversial, as experimental studies have provided conflicting results.

Systemic corticosteroids, although potentially helpful in reducing acute inflammation, are not useful in the treatment of chronic ICD unless corrective measures are taken to avoid the offending contactants.
Narrowband ultraviolet B or photochemotherapy

(PUVA) irradiation may be considered for chronic dermatitis that does not respond to any other form of therapy.

Hyperkeratotic palmoplantar dermatitis from frictional or chronic ICD or a combination of dermatitis and psoriasis may benefit from the adjunctive use of systemic retinoids such as acitretin and alitretinoin or systemic immunomodulators such as methotrexate, cyclosporine, and possibly targeted (biologic) therapy.
Allergic Contact Dermatitis Clinical Presentation
Evaluation of allergic contact dermatitis requires a much more detailed history than most other dermatologic disorders.

Awareness of current trends is also important. The increased use of face masks in the coronavirus disease 2019 (COVID-19) pandemic has been associated with contact dermatitis due to formaldehyde releasers.
Preexisting skin diseases

Individuals with stasis dermatitis are at high risk for developing allergic contact dermatitis to materials and agents applied to the areas of stasis dermatitis and leg ulcers. Neomycin and bacitracin are important causes of allergic contact dermatitis in these individuals because they are used frequently despite the lack of documentation of their efficacy in the treatment of stasis ulcers.
- Atopic dermatitis
- Patients with a history of atopic dermatitis are at increased risk for developing nonspecific hand dermatitis and irritant contact dermatitis.
Onset of symptoms

Individuals with allergic contact dermatitis typically develop dermatitis, within a few days of exposure, in areas that were exposed directly to the allergen. Certain allergens (eg, neomycin) penetrate intact skin poorly, and the onset of dermatitis may be delayed up to a week following exposure.
The immediate onset of dermatitis following initial exposure to material suggests either a cross-sensitization reaction, prior forgotten exposure to the substance, or nonspecific irritant contact dermatitis provoked by the agent in question.
Eyelid dermatitis

Individuals may develop dermatitis on eyelids and other exposed skin following exposure to airborne allergens or allergens transferred to that site by the fingers. Contact dermatitis may also result from allergy to eyelid makeup.
Latex

Rubber latex currently is the most important source of allergic contact urticaria. The term hypoallergenic may refer to gloves that do not contain sensitizing chemicals added to rubber latex but may not indicate whether the gloves are rubber latex free.
Individuals with hand dermatitis, hospital workers, children with spina bifida, and atopic individuals are at increased risk of developing contact urticaria to rubber latex.

Individuals may have allergic contact dermatitis to chemicals added to rubber gloves and have contact urticaria to latex. Individuals wearing rubber gloves should be evaluated carefully for both possibilities.
The hands are the sites exposed most intensely to contact allergens and irritants, both at work and at home. Allergic contact dermatitis in response to workplace materials may improve initially on weekends and during holidays, but individuals with chronic dermatitis may not demonstrate the classic history of weekend and holiday improvement.
Medications

Patients with dermatitis that does not clear with topical corticosteroid treatment should be considered for patch testing with a corticosteroid series and the commercial preparations of corticosteroids and their vehicles.
Physical Examination

Acute allergic contact dermatitis is characterized by pruritic papules and vesicles on an erythematous base. Lichenified pruritic plaques may indicate chronic allergic contact dermatitis. Occasionally, allergic contact dermatitis may affect the entire integument (ie, erythroderma, exfoliative dermatitis). The initial site of dermatitis often provides the best clue regarding the potential cause of allergic contact dermatitis.
SEBORRHEIC DERMATITIS
Seborrheic dermatitis is a common mild chronic eczema typically confined to skin regions with high sebum production and the large body folds. Although its pathogenesis is not fully elucidated, there is a link to sebum overproduction (seborrhea) and the commensal yeast *Malassezia*. 
There are infantile and adult forms, with the former being self-limited and confined to the first 3 months of life, while the latter is chronic with a peak in the fourth to sixth decades.

The prevalence of seborrheic dermatitis is estimated to be 5%.

Extensive and therapy-resistant seborrheic dermatitis is an important cutaneous sign of HIV infection. It is also more commonly observed in patients with Parkinson disease, cerebrovascular accidents, and mood disorders.
There is no simple quantitative relationship between yeast number and severity of seborrheic dermatitis, and unaffected skin may carry a load of organisms similar to seborrheic dermatitis lesions. Seborrheic dermatitis occurs predominantly in areas of the skin with active sebaceous glands and is often associated with sebum overproduction.
However, patients with seborrheic dermatitis may have normal sebum production and those with excessive sebum production are often free of seborrheic dermatitis. Thus, the amount of sebum produced alone does not appear to be the decisive risk factor.

In patients with seborrheic dermatitis, triglycerides and cholesterol are elevated but squalene and free fatty acids are significantly decreased.

Free fatty acids (which have a known antimicrobial effect) are formed from triglycerides by bacterial lipases, produced by the lipolytic Propionibacterium (Corynebacterium) acnes.

A major constituent of the resident microbial skin flora, P. acnes has been found to be greatly reduced in seborrheic dermatitis.
Clinical Features

- Seborrheic dermatitis is defined by clinical parameters, including: sharply demarcated patches or thin plaques that vary from pink–yellow to dull red to red–brown with bran-like to flaky “greasy” scales; vesication and crusting may occur but are rare and mostly due to irritation.
- a predilection for areas rich in sebaceous glands – scalp, face, ears, presternal region – and, less often, the intertriginous areas.
- mild course with little or moderate discomfort.
- Generalized and even erythrodermic forms can occur, albeit rarely.
Infantile seborrheic dermatitis

- This form usually begins about one week after birth and may persist for several months. Initially, mild greasy scales adherent to the vertex and anterior fontanelle regions arise which may later extend over the entire scalp.

- Inflammation and oozing may finally result in a coherent scaly and crusty mass covering most of the scalp cradle cap.

- Lesions of the axillae, inguinal creases, neck, and retroauricular folds are often acutely inflamed, oozing, sharply demarcated, and surrounded by satellite lesions.

- Superinfection with *Candida* spp. or occasionally bacteria (e.g. group A *Streptococcus*) can occur.
**Adult seborrheic dermatitis**

- In adults, seborrheic dermatitis is generally found on the scalp and, usually of milder intensity, on the face; less often, lesions occur on the central upper chest and the intertriginous areas. Erythrodermic seborrheic dermatitis has been described as a rarity.

- In seborrheic dermatitis of the scalp, there is inflammation and pruritus in addition to dandruff.

- The vertex and parietal regions are predominantly affected, but in a more diffuse pattern than the discrete plaques of psoriasis.
Seborrheic dermatitis of the facial skin is often strikingly symmetric, affecting the forehead, medial portions of the eyebrows, upper eyelids, nasolabial folds and lateral aspects of the nose, retroauricular areas, and occasionally the occiput and neck.

Seborrheic dermatitis, like inverse psoriasis, is a cause of intertrigo.

In patients with seborrheic dermatitis, the skin is sensitive to irritation, and exposure to sun or heat, febrile illnesses, and overly aggressive topical therapy may precipitate flares and dissemination.
Malassezia

(Pityrosporum) folliculitis is another complication characterized by pruritic erythematous follicular papules, sometimes pustules, typically in sites rich in sebaceous glands.

The facial immobility of patients with Parkinson disease might result in a greater accumulation of sebum on the skin, resulting in a permissive effect on the growth of Malassezia.

Rebound flares of seborrheic dermatitis can follow tapers of systemic corticosteroids.
Treatment

Infantile seborrheic dermatitis

Infantile seborrheic dermatitis usually responds satisfactorily to bathing and application of emollients. Ketoconazole cream (2%) is indicated in more extensive or persistent cases.

Short courses of low-potency topical corticosteroids may be used initially to suppress inflammation.
Adult seborrheic dermatitis

- The mainstay of therapy is the use of topical azoles (e.g. ketoconazole), either as shampoos (scalp) or as creams (body).
- Ciclopirox olamine has antifungal and anti-inflammatory activities and has also been shown to be effective as a shampoo or cream in double-blind, randomized trials.
- Additional measures, particularly in the initial stages of treatment, include emollients and low-potency topical corticosteroids.
- Second-line treatment options include zinc pyrithione, selenium sulfide, and tar shampoos as well as topical calcineurin inhibitors.
Atopic Dermatitis
Atopic dermatitis (AD) is a chronically relapsing skin disease that occurs most commonly during early infancy and childhood.

It is frequently associated with elevated serum IgE levels and a personal or family history of AD, allergic rhinitis, and/or asthma.

Prevalence in children of 10 to 20 percent in the United States.

The prevalence of AD in adults is approximately 1 to 3 percent.

There is also a female preponderance for AD, with an overall female/male ratio of 1.3:1.
CLINICAL MANIFESTATIONS

- AD typically begins during infancy.
- Approximately 50 percent of patients develop this illness by the first year of life and an additional 30 percent between the ages of 1 and 5 years.
- Nearly 80 percent of patients with AD eventually develop allergic rhinitis or asthma later in childhood.
- Intense pruritus and cutaneous reactivity are cardinal features of AD.
- Its consequences are scratching, prurigo papules, lichenification, and eczematous skin lesions.
Infantile phase (0-2 years)

- Majority start within 6 m and onset around 3 m most common but earlier onset unusual.
- Face, scalp, extensors, napkin area rarely affected.

Childhood phase (2-12 years)

- Flexural involvement, antecubital and popliteal.
- Adolescent phase (12-18 years)
- Flexural and upper trunk and eyelids.
DIAGNOSIS

- Major criteria - all required
- Pruritus
- Typical morphology and distribution of rash.
- Common findings (at least two)
- Personal or family history of atopy
- Immediate skin test reactivity
- White dermographism
- Anterior subcapsular cataracts
- Associated findings (at least four)

- Ichthyosis'
xerosis'
hyperlinear palms'
pityriasis alba'
facial pallor'
infraorbital darkening'
dennie-morgan folds'
keratoconus'
hand dermatitis'
repeated cutaneous infection.
Complications

- **OCULAR PROBLEMS** Eyelid dermatitis and chronic blepharitis are commonly associated with AD and may result in visual impairment from corneal scarring.
- Atopic keratoconjunctivitis is usually bilateral and can have disabling symptoms that include itching, burning, tearing, and copious mucoid discharge.
- Keratoconus is a conical deformity of the cornea believed to result from chronic rubbing of the eyes in patients with AD and allergic rhinitis.
- Cataracts were reported in the early literature to occur in up to 21 percent of patients with severe AD.
The most serious viral infection is herpes simplex, which can affect patients of all ages, resulting in Kaposi’s varicelliform eruption or eczema herpeticum.

After an incubation period of 5 to 12 days, multiple, itchy, vesiculopustular lesions erupt in a disseminated pattern; vesicular lesions are umbilicated, tend to crop, and often become hemorrhagic and crusted. Punched out and extremely painful erosions result. These lesions may coalesce to large, denuded and bleeding areas that can extend over the entire body.

Superficial fungal infections are also more common in atopic individuals and may contribute to the exacerbation of AD. Patients with AD have an increased prevalence of *Trichophyton rubrum* infections compared to nonatopic controls.

As discussed earlier, *S. aureus* is found in more than 90 percent of AD skin lesions. Honey-colored crusting, folliculitis, and pyoderma are indicators of secondary bacterial skin infection, usually due to *S. aureus* that requires antibiotic therapy.
HAND DERMATITIS  Patients with AD often develop a nonspecific, irritant hand dermatitis.

It is frequently aggravated by repeated wetting and by washing of the hands with harsh soaps, detergents, and disinfectants.

Atopic individuals with occupations involving wet work are prone to develop an intractable hand dermatitis in the occupational setting. This is a common cause of occupational disability.
**EXFOLIATIVE DERMATITIS**  Patients with extensive skin involvement may develop exfoliative dermatitis.  

This is associated with generalized redness, scaling, weeping, crusting, systemic toxicity, lymphadenopathy, and fever.  

Although this complication is rare, it is potentially life-threatening.  

It is usually due to superinfection, for example, with toxin-producing *S. aureus* or herpes simplex infection, continued irritation of the skin, or inappropriate therapy.  

In some cases, the withdrawal of systemic glucocorticoids used to control severe AD may be a precipitating factor for exfoliative erythroderma.
Infiltrated, erythematous facial lateral thinning of eyebrows and infraocular (Morgan's) fold
DIFFERENTIAL DIAGNOSIS

- Of the major features, pruritus and chronic or remitting eczematous dermatitis with typical morphology and distribution are essential for diagnosis.
- Lists a number of inflammatory skin diseases, immunodeficiencies, skin malignancies, genetic disorders, infectious diseases, and infestations that share symptoms and signs with AD.
Infants presenting in the first year of life with failure to thrive, diarrhea, a generalized scaling erythematous rash, and recurrent cutaneous and/or systemic infections should be evaluated for severe combined immunodeficiency syndrome.

Wiskott-Aldrich syndrome is an X-linked recessive disorder characterized by cutaneous findings almost indistinguishable from AD. It is associated with thrombocytopenia, variable abnormalities in humoral and cellular immunity, and recurrent severe bacterial infections.

Hyperimmunoglobulin-E syndrome is characterized by markedly elevated serum IgE levels, defective T cell function, recurrent deep-seated bacterial infections, including cutaneous abscesses due to *S. aureus* and/or pruritic skin disease due to *S. aureus* pustulosis, or by recalcitrant dermatophytosis.
It is important to recognize that an adult who presents with an eczematous dermatitis with no history of childhood eczema, respiratory allergy, or atopic family history may have allergic contact dermatitis.

A contact allergen should be considered in any patient whose AD does not respond to appropriate therapy.

Of note, topical glucocorticoid contact allergy has been reported increasingly in patients with chronic dermatitis on topical corticosteroid therapy.

Cutaneous T cell lymphoma must be ruled out in any adult presenting with chronic dermatitis poorly responsive to topical glucocorticoid therapy.
Eczematous dermatitis has been also reported with HIV as well as with a variety of infestations such as scabies.

Other conditions that can be confused with AD include psoriasis, ichthyoses, and seborrheic dermatitis.
Keratolysis exfoliativa.
Irritant hand dermatitis
Nummular eczema.
CUTANEOUS HYDRATION

TOPICAL GLUCOCORTICOID TREATMENT

Topical Immunomodulators

*Tacrolimus*

Tacrolimus inhibits the activation of a number of key cells involved in AD including T cells, Langerhans cells, mast cells, and keratinocytes.

*Pimecrolimus*

INFECTIOUS AGENTS

Phototherapy

Ultraviolet B (311 nm), UVA-1 (340 to 400 nm), and combined UVAB phototherapy can be useful adjuncts in the treatment of AD.

**CYCLOSPORINE**
- **ANTIMETABOLITES** Mycophenolate mofetil (MMF).
- Methotrexate.
- Azathioprine.
- **EXTRACORPOREAL PHOTOPHERESIS**
Following predictive factors correlate with a poor prognosis for AD: widespread AD in childhood; associated allergic rhinitis and asthma; family history of AD in parents or siblings; early age at onset of AD; being an only child; and very high serum IgE levels.
Prurigo Nodularis
Prurigo nodularis is an uncommon disease of unknown cause that may be considered a nodular form of lichen simple chronicus.

Few to 20 or more nodules are randomly distributed on the extensor aspects of the arms and legs.

They are created by repeated scratching. The nodules are red or brown, hard, and dome-shaped with a smooth, crusted, or warty surface; they measure 1 to 2 cm in diameter.
Hypertrophy of cutaneous papillary dermal nerves is a relatively constant feature.

Complaints of pruritus vary. Some patients claim there is no itching and that scratching is only habitual, whereas others complain that the pruritus is intense.
Treatment. Prurigo nodularis is resistant to treatment and lasts for years

- Repeated intralesional steroid injections
- Excision of individual nodules
- Capsaicin
- The use of combination or sequential topical calcipotriene with topical steroids has been effective.
- Naltrexone (50 mg daily).
- Oral cyclosporine (3 to 5 mg/kg daily) was effective in one study.
- Gabapentin has been used in resistant cases.
- Depression may be associated with this disorder and psychiatric referral may be appropriate.
Stasis Dermatitis
**Stasis Dermatitis**

- **Etiology.** Stasis dermatitis is an eczematous eruption that occurs on the lower legs in some patients with venous insufficiency.

- The dermatitis may be acute, subacute, or chronic and recurrent, and it may be accompanied by ulceration.

- Most patients with venous insufficiency do not develop dermatitis, which suggests that genetic or environmental factors may play a role.

- Some have speculated that it represents an allergic response to an epidermal protein antigen created through increased hydrostatic pressure, whereas others believe that the skin has been compromised and is more susceptible to irritation and trauma.
Allergy to Topical Agents. Patients with stasis dermatitis have significantly more positive reactions when patch tested with components of previously used topical agents.

Topical medications that contain potential sensitizers such as lanolin, benzocaine, parabens, and neomycin should be avoided by patients with stasis disease.

Allergy to corticosteroids in topical medication is also possible.
Types of Eczematous Inflammation

*Subacute Inflammation*

- Subacute inflammation usually begins in the winter months when the legs become dry and scaly.
- Brown staining of the skin (hemosiderin) may have appeared slowly for months.
- The pigment is iron remaining after disintegration of red blood cells that leaked out of veins because of increased hydrostatic pressure.
- Scratching induces first subacute and then chronic eczematous inflammation.
**Acute Inflammation**

- A red, superficial itchy plaque may suddenly appear on the lower leg. This acute process may be eczematous inflammation, cellulitis, or both.
- Weeping and crusts appear. A vesicular eruption (id reaction) on the palms, trunk, and/or extremities sometimes accompanies this acute inflammation.
- The inflammation responds to systemic antibiotics, wet compresses, and group III to V topical steroids.
- Wet compresses should be discontinued before excessive drying occurs.
- The id reaction resolves spontaneously as the primary site improves.
**Chronic Inflammation**

- Recurrent attacks of inflammation eventually compromise the poorly vascularized area, and the disease becomes chronic and recurrent.
- The typical presentation is a cyanotic red plaque over the medial malleolus.
- Fibrosis following chronic inflammation leads to permanent skin thickening. The skin surface in these irreversibly changed areas may have a bumpy, cobblestone appearance that results from fibrosis and venous and lymph stasis.
- The skin remains thickened and diffusely dark brown (postinflammatory hyperpigmentation) during quiescent periods.
Treatment of Stasis Dermatitis

Topical Steroids and Wet Dressings. The early, dry superficial stage is managed as subacute eczematous inflammation with group II to V topical steroid creams or ointments and lubricating creams or lotions.

Oral antibiotics (usually those active against Staphylococci, e.g., cephalexin) hasten resolution if cellulitis is present.

Moist exudative inflammation and moist ulcers respond to tepid wet compresses of Burow’s solution or merely saline or water for 30 to 60 minutes several times a day. Wet dressings suppress inflammation while debriding the ulcer.
Adherent crust may be carefully freed with blunt-tipped scissors.

Group V topical steroids are applied to eczematous skin at the periphery of the ulcer. Patients must be warned that steroid creams placed on the ulcer stop the healing process.

Elevation of the legs encourages healing.
Severe, painful, exudative, weeping infected eczema with moist crust.
red, itchy plaque may suddenly develop acute inflammation