

Mimics of Dermatitis



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KEYWORDS

- Atopic dermatitis (AD) • Allergic contact dermatitis (ACD) • Psoriasis
- Dermatophytosis • Scabies • Cutaneous T-cell lymphoma (CTCL)
- Subacute cutaneous lupus erythematosus (SCLE) • Dermatomyositis (DM)

KEY POINTS

- The diagnosis of dermatitis can be challenging because many skin diseases can mimic dermatitis.
- It is important to recognize and be familiar with the skin diseases that can resemble dermatitis because some of them can represent signs of systemic disease, infection, infestation, or malignancies.
- Thorough history and physical examination, skin biopsy, and diagnostic tests are essential for establishing an accurate diagnosis and providing optimal care for the patient.

INTRODUCTION

Dermatitis is a common condition frequently encountered by dermatologists. The diagnosis of dermatitis can be challenging because this condition is often multifactorial, and many skin diseases that can mimic dermatitis should be considered in the differential diagnosis.¹ When a patient presents with a pruritic, crusty, eczematous, and erythematous rash in a widespread distribution, clinicians most frequently consider the diagnosis of atopic dermatitis (AD) or allergic contact dermatitis (ACD), which are common conditions in dermatology. However, it is important to recognize and be familiar with other skin diseases that can resemble dermatitis but represent signs of systemic disease or malignancies because misdiagnosis can lead to mismanagement and adverse outcomes for the patient. What follows is a discussion of some of the diagnoses to consider when evaluating a patient with dermatitis.

DERMATITIS ASSOCIATED WITH ALLERGY

Atopic Dermatitis

AD is a chronic, pruritic, inflammatory skin condition characterized by a remitting and relapsing disease course.² AD occurs most frequently in children, with about 20% prevalence, and historically has been viewed as a pediatric disease, with 50% of

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Immunol Allergy Clin N Am 41 (2021) 493–515

<https://doi.org/10.1016/j.iac.2021.04.009>

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atopic children developing this condition in the first year of life and 85% by 5 years of age.³⁻⁶ However, AD is not only a disease of childhood but also affects many adults. It can persist into adulthood or present as adult-onset disease (Fig. 1A, B).⁷ Prevalence of AD in adults in Western industrialized countries is reported to be around 5%.^{8,9} This disorder is linked to many comorbidities and, in addition to other atopic march conditions, AD has been associated with increased reports of neuropsychiatric, behavioral, cardiovascular, and malignant diseases, suggesting that it should be regarded as systemic disorder.^{10,11} The impact of the financial burden of this chronic condition on patients and society is substantial, with an estimated annual cost of \$5.297 billion dollars in the United States in the year 2015.¹² The clinical presentation of AD varies with age and ethnicity.^{3,13} Intensely pruritic, eczematous lesions accompanied by oozing, weeping, and crusting are common on the scalp, face, neck, trunk, and extensor extremities of infants, usually sparing the diaper area. In childhood, dry, erythematous scaly plaques are common in flexural sites such as the antecubital and popliteal fossa, neck creases, ventral wrists, and ankles.^{2,8} Adults with AD have heterogenous clinical presentations, with higher prevalence of hand-and-foot dermatitis (Fig. 2A, B), nummular eczema, head-and-neck dermatitis, hyperlinear palms, erythroderma, generalized xerosis, lichenified skin changes, and prurigo nodules.^{13,14} Because of diverse clinical presentations of adult AD, which differs from the classic flexural pattern of childhood disease, and lack of standardized diagnostic criteria, AD in adults should be regarded as a diagnosis of exclusion and other dermatologic conditions that can mimic AD should be carefully considered.¹⁴ Adult-onset dermatitis may be over-diagnosed, and according to recent studies about 1 in 4 adults with AD self-report adult-onset disease.³ The differential diagnosis of AD is extensive (Table 1) and includes inflammatory dermatoses, infestations, infections, immune-mediated conditions, and malignant diseases.^{2,15,16} Good history, thorough physical examination, skin biopsy, and diagnostic tests are essential for establishing an accurate diagnosis and providing optimal care for the patient.

Allergic Contact Dermatitis

A common differential diagnosis to AD is ACD.

ACD is an inflammatory skin condition caused by a delayed type IV hypersensitivity reaction in response to an allergen or allergens. The first step in the disease process is exposure to a chemical substance that results in a cascade of immune responses

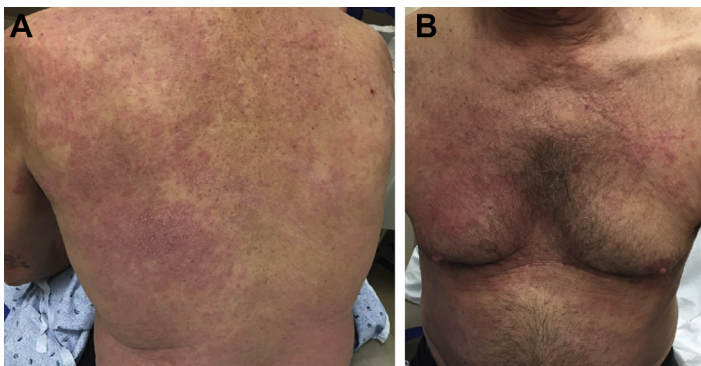


Fig. 1. (A, B) Adult-onset AD: erythematous, dry, and scaly plaques located on the back, chest, and abdomen of middle-aged white man.



Fig. 2. (A, B) Hand dermatitis: erythema with scaling, crusting, and erosions involving the fingers and extending onto the palmar surface of hands.

resulting in sensitization. On repeat exposure in the sensitized individual, elicitation occurs with subsequent dermatitis.¹⁷ ACD has a spectrum of clinical presentations. The acute phase is characterized by severe pruritus, poorly demarcated erythema, and edema expanding beyond the contact area as well as microvesiculations or, in severe cases, even bullae formation. In the subacute phase, scaly and crusty lesions develop with possible erosions. The chronic phase is characterized by less exudative lesions with features of lichenification, scaling, and fissuring.^{17–19}

Acute ACD develops within 24 to 72 hours after allergen reexposure and initially presents with asymmetric eruption limited to the area of contact with the skin. However, it can soon become generalized, mimicking many dermatologic conditions, including AD and making diagnosis more challenging.¹⁹ Therefore, the focus here is on differentiating clinical patterns of ACD from AD.

ACD presents on the face (including eyelids and perioral area), neck, hands, and feet; less frequently it can be seen on the trunk as a patchy generalized distribution and on the scalp. Patient populations at risk of ACD include those with occupational exposures, such as health care workers, beauticians, hair stylist, nail salon workers, auto mechanics, florists, construction workers, and industrial workers. Patch testing is a gold standard in the diagnosis of ACD.²⁰ Biopsy for histopathologic analysis to differentiate between ACD and AD is not helpful because both conditions look similar on hematoxylin-eosin and show spongiosis and superficial perivascular infiltrate with eosinophils.¹⁷ Thus, clinical patterns, especially regional patterns, can provide a diagnostic clue and help distinguish ACD from AD and other dermatoses.

ACD of the face is most commonly caused by personal hygiene and cosmetic products (Fig. 3A–C).²¹ Presence of eczematous plaques in the frontal hairline, on the sides of the face (sideburns) extending onto the lateral neck, and on the posterior neck can be consistent with allergens in hair dye, chemical perms, shampoos, or other hair products. The distribution of rash is caused by the product being applied to the scalp and subsequently rinsed off, leading to rash in the aforementioned distribution. The facial skin is more prone to ACD than skin of the scalp.²²

Dermatitis of the central face presenting in bilateral patchy pattern can be caused by makeup, facial moisturizers, face washes, or gold being released from gold jewelry such as rings, watches, or bracelets reaching the face via the hands.²³ ACD of the eyelids is very common owing to frequent allergen exposure. However, as detailed previously, AD is also common in this location, as well as other skin conditions such as seborrheic dermatitis, psoriasis, and dermatomyositis (DM).²⁴ When eczematous

Table 1
Mimics of dermatitis

Differential Diagnosis	Age of Onset	Distribution	Morphologic Features
Psoriasis	Children and adults Bimodal age distribution in adulthood, with first peak around 20–30 y of age and second peak at 50–60 y of age	Classically: elbows, knees, extensor surfaces of the forearms and shins, umbilicus, intragluteal cleft, scalp, and nails Other common locations: face (upper and lower forehead, periauricular area, ears, and cheeks), intertriginous/ flexural areas, genitals, palms, soles, generalized (erythroderma)	Sharply demarcated oval, round, or irregular pink to erythematous plaques covered with thick, white to silvery scale. Lesions of psoriasis are dry, often causing fissuring and bleeding (Auspitz sign)
Dermatophyte infection	Children and adults	Scalp (tinea capitis), face (tinea faciei), beard area (tinea barbae), hands (tinea manuum), body (tinea corporis), groin (tinea cruris), feet (tinea pedis), and nails (tinea unguium)	Single or multiple red, annular or oval, scaly plaque varying in size with slightly raised active border and central clearing. Borders of the plaque have leading scale and advance centrifugally
Scabies	Children and adults	Infants and children: palms, soles, face, neck, and scalp Adults: interdigital web spaces, flexural surfaces of the wrists, axillae, belt line, periumbilical skin, groin, buttocks, periareolar areas in women and penile shaft in men	Presence of burrows is pathognomonic, but can be difficult to detect because of secondary excoriations and crusting from frequent impetiginization Intense nocturnal pruritus, urticarial papules, nodules, and vesicles represent the signs of host hypersensitivity reaction to the mite
Cutaneous T-cell lymphoma	Adults: between 50 and 60 y of age, M>F Children: rare	Non-sun-exposed area or a bathing-suit distribution, which includes chest, lower trunk, buttocks, and groin	Classic morphologies are patch, plaque, and tumor stage Early disease: well-defined, scaly erythematous patches or thin plaques with atrophic surface Disease slowly progresses and can become widespread, and advanced stage characterized by infiltrative plaques, cutaneous tumors, erythroderma, and eventually nodal and visceral involvement

Subacute cutaneous lupus erythematosus	Adults: between 20 and 40 y of age, F>M	Sun-exposed areas of lateral face, V area of the neck, upper back, shoulders, and extensor surfaces of the arms	Photodistributed, symmetric, nonscarring, erythematous macules and papules that evolve into either papulosquamous, psoriasiform plaques or scaly annular/polycyclic plaques with confluence, central hypopigmentation, and clearing
Dermatomyositis	Children: between 4 and 14 y of age Adults: between 40 and 60 y of age, F>M	Gottron papules: dorsal surface of metacarpophalangeal and interphalangeal joints Gottron sign: extensor tendons of dorsal hands in a linear fashion, dorsal-lateral sides of finger joints, elbows, knees, and medial malleoli Heliotrope rash: periorbital, more pronounced on the upper eyelids The shawl sign: posterior shoulders, upper back, and neck The V sign: anterior neck and upper chest The holster sign: lateral upper thighs and hips Other areas: scalp, nailfolds, and hands (mechanic's hands)	Gottron papules: lichenoid, violaceous, flat-topped papules and thin plaques with slight atrophy and subtle scaling Gottron sign: symmetric, slightly scaly erythematous to violaceous macules and patches Heliotrope rash: symmetric periorbital violaceous erythema with edema The shawl sign and the V sign: poikilodermatous skin changes (hypopigmentation or hyperpigmentation, telangiectasia, and atrophy) with macular violaceous erythema in photoexposed areas Symmetric poikiloderma Scalp: erythema, scale, and atrophy Nailfolds: periungual erythema with telangiectasia of the proximal nail folds, dystrophic and ragged cuticles (Samitz sign), dilated capillary loops with dropout Mechanic's hands: hyperkeratosis, scaling, and fissuring of the fingertips, palms, and lateral aspects of the first, second, and third digits

Abbreviations: F, female; M, male.



Fig. 3. (A, B) ACD of the face: erythematous, edematous plaque with small vesicles involving the face with sharp demarcation at the frontal hairline. (C) ACD to benzoyl peroxide.

plaques are located primarily on the lower eyelids extending down onto the cheeks in a linear, driplike pattern, eye drops should be suspected as a cause. Dermatitis involving both upper and lower eyelids in bilateral periorbital pattern can be caused by application of cosmetic products around the eyes. Importantly, an external allergen should be suspected when there is a unilateral eyelid dermatitis, which likely points toward transmission of an allergen from the hands.²²

Dermatitis of the hands is very common. It is hard to differentiate chronic contact dermatitis of the hands based on morphology or histology from AD, dyshidrotic eczema, or palmoplantar nonpustular psoriasis.²⁵ Detailed history, including patient occupation and hobbies, followed by patch testing is extremely important if ACD is suspected. Dermatitis involving a tip of the thumb and the medial sides of the index

finger and middle finger of the dominant hand in a pincer grasp pattern has been reported in florists from *Alstroemeria*, the Peruvian lily, *Chrysanthemum*, and *Hydrangea*.^{22,26–28} Periungual eczematous dermatitis has been linked to nail cosmetics, specifically acrylates, and the number of cases have been increasing with increased use of photobonded acrylic gel nails.²⁹ Contact dermatitis from acrylic nails can also present on the face from airborne transfer of acrylic dust or direct transfer from the hands.

Foot dermatitis can result from the exposure to the allergens present in shoes, such as rubber, leather, and adhesives.³⁰ ACD of the foot can be mistaken for tinea pedis, psoriasis, dyshidrotic eczema, AD, and juvenile plantar dermatosis. Foot dermatitis caused by footwear usually occurs on the dorsum of the foot in bilateral distribution with sparing of the web spaces.²² It is important to perform a scraping for potassium hydroxide (KOH) to rule out dermatophyte infection. If allergy is suspected, referral for patch testing is necessary.

Psoriasis

Psoriasis is a common chronic, inflammatory, immune-mediated disease affecting about 2% to 3% of the general population.³¹ Psoriasis affects both genders equally and can present at any age. However, bimodal age distribution is commonly observed, with the first peak around 20 to 30 years of age and a second peak at 50 to 60 years of age.³² Psoriasis is a systemic inflammatory disorder and its involvement extends well beyond the skin. This condition is associated with multiple comorbidities, including psoriatic arthritis, cardiovascular disease, metabolic syndrome, gastrointestinal diseases, kidney disease, malignancy, and mood disorders.³³ It represents significant physical and socioeconomic burden and negatively affects quality of life in affected individuals.

Plaque psoriasis is the most common type.³⁴ It typically presents as sharply demarcated oval, round, or irregular pink to erythematous plaques covered with thick, white to silvery scale (Fig. 4). Lesions of psoriasis are dry, often causing fissuring and bleeding. They can cause pruritus as well as pain and burning sensation. Common locations include elbows, knees, extensor surfaces of the forearms (Fig. 5A, B) and shins, umbilicus, intragluteal cleft, and the scalp. Nail involvement is also common, affecting approximately 25% of patients with psoriasis.³⁴

The differential diagnosis of psoriasis is vast and can include AD, nummular eczema, lichen simplex chronicus, pityriasis rosea, and tinea.³⁵ When clinical findings are distinctive with sharply demarcated, scaly erythematous plaques distributed symmetrically on extensor extremities, the diagnosis is straightforward. However, there are several variants of psoriasis, including atypical presentations that can mimic dermatitis and cause difficulty in diagnosis.

Facial psoriasis affects about 20% of patients with psoriasis vulgaris and is more commonly associated with longer disease duration and family history of psoriasis.³⁶ Clinical presentation is characterized by sharply demarcated, red, scaly, symmetric plaques associated with itching, soreness, and increased sensitivity. The most commonly involved areas of the face include upper forehead (76%), lower forehead (52%), periauricular area (46%), ears (39%), and cheeks (39%).³⁷ Psoriasis does not tend to affect eyelids, perioral area, or nasolabial folds.

Scalp psoriasis occurs in 45% to 56% of patients with psoriasis.³⁷ It can be easily confused with seborrheic dermatitis or tinea capitis. Lesions of scalp psoriasis are well-demarcated xerotic plaques with silvery-white flaky scale.³⁸ Plaques of scalp psoriasis are commonly located at the occiput and may advance beyond the border of the hairline. Affected areas of the scalp are extremely dry and are associated with cracking



Fig. 4. Plaque psoriasis: sharply demarcated pink to erythematous plaques covered with scale.

and bleeding. Patients usually complain of pain, soreness, and burning sensation. Pruritus is a hallmark feature of scalp psoriasis that leads to scratching and picking.

Inverse psoriasis, also known as intertriginous or flexural psoriasis, affects 20% to 30% of patients with psoriasis. It involves the axillae, inguinal folds, intergluteal cleft,



Fig. 5. (A, B) Psoriasis: sharply demarcated pink, scaly plaque involving the elbow, extensor surface of the forearm, and extending onto the dorsal hand and fingers.

neck folds, groin and external genitalia, umbilicus, postauricular area, and antecubital and popliteal fossae.³⁹ Skin lesions of inverse psoriasis are well-demarcated brightly erythematous smooth plaques that lack significant scale. They appear shiny and glazed because of the soft and moist flexural environment. Superficial erosion and maceration are common because of sweating and friction and can cause irritation and soreness.³⁸

Palmoplantar psoriasis accounts for about 14% of cases.³⁴ This form of psoriasis is arguably one of the most debilitating and causes high morbidity and severe pain. It usually presents between 30 and 40 years of age and affects mostly women, with female/male ratio of 9:1.⁴⁰ Hyperkeratotic and pustular variants exist. The hyperkeratotic phenotype presents with red, dry, and thickened palms and soles with deep painful fissures.³⁶ The pustular variant presents with recurrent eruption of small sterile pustules and erythema on the palmar and plantar surfaces.³⁷ Diagnosis of palmoplantar psoriasis is challenging because of the clinical overlap with chronic ACD, dyshidrotic eczema, and AD with hand involvement.

Erythrodermic psoriasis is a very rare, potentially life-threatening form of psoriasis, affecting 1% of patients with psoriasis.⁴⁰ Erythroderma is an inflammatory skin syndrome that presents as generalized erythema and desquamation of more than 90% of the body surface area.⁴¹ It is associated with high mortality because of loss of thermoregulation and skin barrier function that can lead to fever, hypothermia, fluid loss, electrolyte imbalance, dehydration, and shock.⁴⁰ Psoriasis is the most frequent cause of erythroderma, representing 25% to 50% of cases. However, other causes of generalized erythroderma include AD, drug reactions, cutaneous T-cell lymphoma (CTCL), pityriasis rubra pilaris, DM, Langerhans cell histiocytosis, and papuloerythroderma of Ofuji.⁴¹ Distinguishing erythrodermic psoriasis from the other causes of erythroderma can be very challenging.

Dermatophyte Infection

Dermatophytosis or tinea is a superficial fungal infection of the skin, hair, and nails caused by dermatophytes. Three major groups of dermatophytes that cause tinea are *Trichophyton*, *Epidermophyton*, and *Microsporum*.⁴² Dermatophytes are the most common cause of superficial fungal infections in humans, with reported worldwide prevalence of 20% to 25%.⁴³ The most common sites of infection include the scalp (tinea capitis), face (tinea faciei), beard area (tinea barbae), hands (tinea manuum), body (tinea corporis), groin (tinea cruris), feet (tinea pedis), and nails (tinea unguium).⁴⁴

Tinea corporis, colloquially known as body ringworm, involves the trunk, neck, arms, and legs. It classically presents as a red, annular or oval, scaly plaque, with slightly raised active border and central clearing. Borders of the plaque have leading scale and advance centrifugally. Lesions can be single or multiple and vary in size.⁴⁵ Tinea corporis can be easily misdiagnosed as nummular eczema, contact dermatitis or AD, seborrheic dermatitis, psoriasis, or erythema annulare centrifugum.⁴⁶ KOH 10% to 20% preparation applied to skin scrapings from the active margin of the lesion and examined under the microscope help establish the diagnosis of tinea. KOH scrapings are positive when refractile, long, smooth, branching, and septate hyphae are visualized (Fig. 6A–C).⁴²

Tinea manuum is dermatophytosis of 1 or both hands and frequently occurs along with tinea pedis.⁴⁷ It presents as erythematous, dry and scaly, hyperkeratotic palms and is frequently confused with atopic hand dermatitis, irritant dermatitis, ACD, or palmar psoriasis.⁴⁸

When cutaneous dermatophytosis is initially unrecognized, because of its resemblance to dermatitis, it is frequently treated with topical antiinflammatory agents

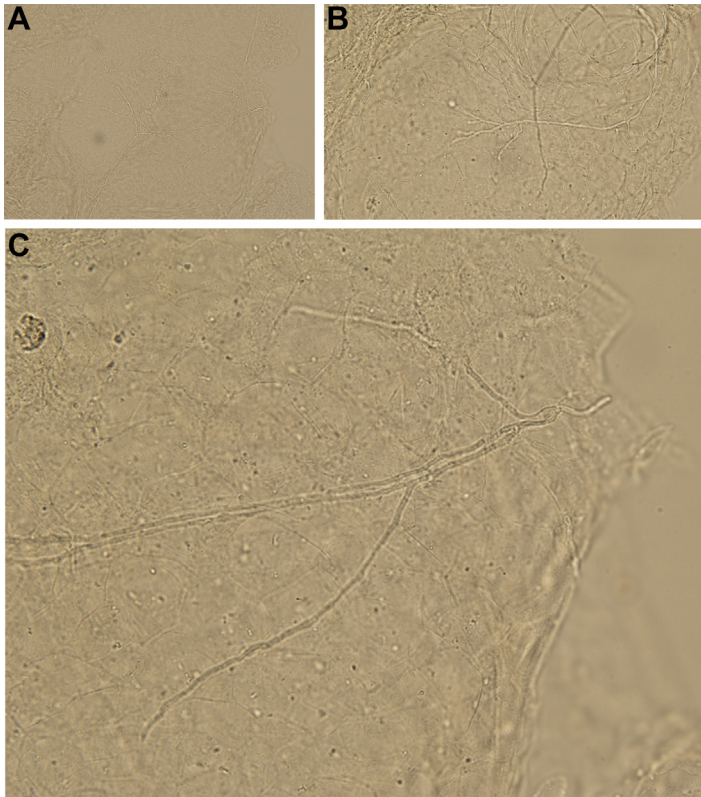


Fig. 6. (A–C) KOH 10% preparation positive for tinea with refractile, long, smooth, branching, and septate hyphae. (A) Original magnification x 200; (B) Original magnification x 400; (C) Original magnification x 400.

such as corticosteroids or calcineurin inhibitors. As a result, the dermatophyte infection loses its typical clinical and morphologic appearance, giving rise to an entity termed tinea incognito (Fig. 7A–C).⁴⁹

Dermatophytosis can progress and become generalized, involving multiple body sites, including face, trunk, hands, and feet. When at least 4 body sites are affected, it is known as trichophyton rubrum syndrome.⁵⁰

Scabies

Scabies is a common, highly contagious, parasitic skin infection caused by the *Sarcoptes scabiei* mite. It is transmitted by close personal contact and indirectly via fomites. The estimated worldwide prevalence of scabies is 300 million cases every year.⁵¹ In developed, industrialized countries, scabies is more frequently observed in the elderly residing in nursing home facilities, as well as in hospitals and overcrowded, impoverished areas. However, in developing countries, scabies is commonly diagnosed in children.⁵² The diagnosis of scabies can be challenging because of the variety of clinical presentations. Scabies can mimic other skin conditions such as AD, allergic and irritant contact dermatitis, psoriasis, diaper dermatitis, drug eruption, and bullous pemphigoid.⁵³ Therefore, a high index of suspicion is needed to make a correct diagnosis. According to a recent study, approximately

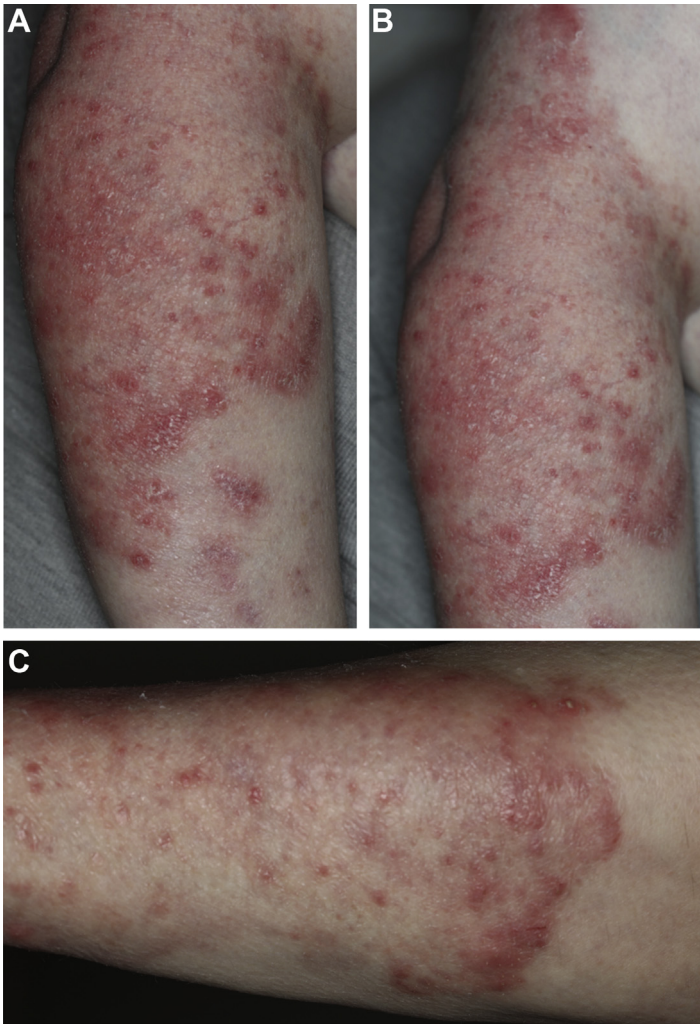


Fig. 7. (A–C) Tinea corporis treated with topical steroids (tinea incognito): erythematous, scaly plaques with partial central clearing and slightly raised active boarder with scattered papules and pustules.

45% of the patients presenting to the dermatology office with scabies had been misdiagnosed by another provider.^{51,54}

Scabies infestation occurs when an adult female mite tunnels through the stratum corneum of the epidermis and lays 2 to 3 eggs daily. This activity results in a host hypersensitivity reaction to the mite invasion leading to signs and symptoms of infestation, including intense nocturnal pruritus, urticarial papules, nodules, and vesicles.⁵⁵ Detection of burrows is pathognomonic; however, it can be difficult because of secondary excoriations and crusting from frequent impetiginization.⁵⁶ In adults, common sites of burrows are interdigital web spaces, flexural surfaces of the wrists, axillae, belt line, periumbilical skin, groin, buttocks, periareolar areas in women, and penile shaft in men. In infants and young children, the palms, soles, face, neck, and scalp are frequently involved.⁵⁷

The confirmation of the diagnosis can be made by obtaining a skin scraping of the burrow with a sterile #15 scalpel blade, placing the contents on a glass slide and applying a drop of mineral oil or 10% KOH preparation to the slide. Visualization of the mite, eggs, or mite's feces (scybala) under the microscope establishes the diagnosis. Because of the low sensitivity of this technique, it is strongly recommended to repeat the scraping in different areas.⁵⁸ Dermoscopy can be a useful tool for identification of the mite at the end of the burrow, which resembles a jetliner with contrail.⁵⁹

Atypical clinical appearance, which further delays and complicates diagnosis of scabies, commonly occurs in patients on topical or systemic steroids (scabies incognita),⁶⁰ in immunocompromised patients with human immunodeficiency virus, as well as cognitively impaired elderly patients (crusted scabies)⁶¹ and those with secondary bacterial infection and long-standing infestation where severe eczematous skin changes predominate.⁶²

Cutaneous T-cell Lymphoma

Mycosis fungoides (MF) and Sezary syndrome (SS) are the most common forms of CTCL, comprising more than half of the cases. The overall incidence of MF/SS is approximately 4 per 1 million people.⁶³ MF usually affects older patients between 50 and 60 years of age; however, it can be seen in younger patients, including children. Men are more commonly affected. MF is seen more frequently in African Americans compared with white people.⁶⁴ Patients diagnosed with CTCL have an increased risk of developing Hodgkin lymphoma and other nonhematologic malignancies.⁶⁵

MF and SS are challenging to diagnose, especially in the early stages of the disease, because of numerous variants and clinical appearances mimicking many benign inflammatory dermatoses, such as eczema, psoriasis, nonspecific generalized dermatitis, and erythroderma.^{65,66}

In classic MF, 3 clinical morphologies (patch, plaque, and tumor) are recognized (Fig. 8A–C).⁶⁴ Early disease presents as well-defined, scaly, erythematous patches or thin plaques with atrophic surface in a non-sun-exposed area or a bathing-suit distribution, which includes the chest, lower trunk, buttocks, and groin.⁶⁵ Early-stage MF is indolent and very slowly progressive disease that can evolve over many years to decades to more widespread and advanced stages characterized by infiltrative plaques, cutaneous tumors, erythroderma, and eventually nodal and visceral involvement.⁶⁷

MF is commonly referred to as a great imitator because of its wide clinicopathologic variants that mimic inflammatory conditions.⁶⁸ Such MF variants include, but are not limited to, hypopigmented, ichthyosiform, psoriasiform, and MF palmaris et plantaris, which contribute to the difficulty in diagnosing this disease.

Hypopigmented MF more commonly affects the pediatric population and skin of patients of color. It presents as round, oval, or irregular nonatrophic hypopigmented patches covered by fine scale.⁶⁹ Lesions are usually asymptomatic; however, sometimes they can be slightly pruritic. Hypopigmented lesions are typically distributed on the trunk, thighs, buttocks, and extremities.⁷⁰

Ichthyosiform MF is characterized by widespread, dry, scaling patches and thin plaques with comedolike lesions and follicular keratotic papules involving the trunk and more commonly the extremities and resembling an ichthyosis vulgaris-like eruption.^{69,71} This MF type is common in young patients and carries a relatively favorable prognosis.⁷²

Psoriasiform MF presents with thick, scaly, well-demarcated, pink to erythematous psoriasiform plaques characteristic of psoriasis vulgaris. It is important to distinguish between these two conditions because systemic immunosuppressors used for treatment of psoriasis can exacerbate MF and possibly lead to accelerated progression of the disease.⁷¹



Fig. 8. (A) MF: thin, annular, eczematous plaque with erythematous boarder and atrophic center. (B) MF, plaque stage: annular, erythematous, alopecic plaque covered with fine scale on the forearm of white man. (C) MF, tumor stage: red-brown, eczematous, crusty, and scaly plaque with induration and nodularity involving upper vermilion and cutaneous lip of middle-aged white man.

MF palmaris et plantaris is a rare variant of MF that involves the palms and soles with occasional extension onto the dorsal aspect of feet, fingers, and wrists. Typical lesions are bilateral, annular, erythematous, hyperkeratotic, and sometimes hyperpigmented patches and plaques with fissures, scale, verrucous changes, ulcerations, and nail dystrophy.^{69,71} Clinical differential diagnosis includes allergic and/or irritant contact dermatitis, dyshidrotic eczema, and palmoplantar psoriasis.⁶⁹

SS is defined as an aggressive and leukemic variant of CTCL that is characterized by at least 1000 circulating atypical lymphocytes in the peripheral blood and erythroderma accompanied by severe generalized pruritus with or without lymphadenopathy.^{63–66} Generalized exfoliative erythroderma can also present with keratoderma and fissures of the palms and soles.⁶⁵

Subacute Cutaneous Lupus Erythematosus

Subacute cutaneous lupus erythematosus (SCLE) is a subtype of cutaneous lupus erythematosus with distinct clinical presentation and serologic markers. Middle-aged women have much higher incidence of SCLE compared with men, typically with the

disease onset in the third or fourth decade of life.⁷³ Approximately 25% to 30% of patients with SCLE are at risk of developing systemic lupus erythematosus; however, the systemic disease tends to be milder and is mostly limited to joint involvement.⁷⁴ Anti-Ro/SSA antibodies are detected in more than 70% of patients with SCLE and are the most common serologic association.⁷⁵ Secondary Sjögren syndrome is the most common autoimmune connective tissue disorder to overlap with SCLE.⁷⁶ SCLE is characterized by its high degree of photosensitivity, with ultraviolet (UV) light being one of the most common triggers.^{75,77} Other environmental factors that can precipitate or worsen SCLE are cigarette smoking and certain medications, such as thiazide diuretics, calcium channel blockers, proton pump inhibitors, and allylamine antifungals.^{78–80}

The skin lesions of SCLE are photodistributed, symmetric, nonscarring, erythematous macules and papules that evolve into either papulosquamous, psoriasiform plaques or scaly annular/polycyclic plaques with confluence, central hypopigmentation, and clearing (Fig. 9).⁸¹ Sun-exposed areas of the lateral face, V area of the neck, upper back, shoulders, and extensor surfaces of the arms are frequently involved sites.⁸² Clinically, SCLE eruption can be easily mistaken for annular lesions of psoriasis, tinea corporis, AD, nummular eczema, CTCL, or drug reaction.

Photoprovocation with UVA and/or UVB light can help confirm the diagnosis of SCLE. However, it might take up to 3 weeks for UV-induced eruption to occur.⁷⁶ Skin biopsy for histologic evaluation and direct immunofluorescence can also help support the diagnosis. Laboratory test for anti-Ro/SSA and anti-La/SSB antibodies should be performed. Previous studies showed that SCLE can present as paraneoplastic phenomenon associated with hematologic and solid organ malignancy.^{74,83}

Dermatomyositis

DM is a chronic, idiopathic, multisystem autoimmune condition that mainly affects skin and skeletal muscle. DM can occur in both children and adults. It has a bimodal age distribution, with 2 peaks occurring between 4 and 14 years of age (juvenile DM) and between 40 and 60 years of age (adult DM).⁸⁴ Similar to many other autoimmune diseases, DM has female predominance, with female/male ratio of 2:1, and it is more common in the African American patient population.⁸⁵ DM is further subdivided into classic DM, with both muscle and skin involvement; amyopathic DM (ADM), which includes pathognomonic skin rash of DM but lacks any clinical or laboratory evidence of



Fig. 9. SCLE: photodistributed, annular, coalescing pink plaques with central hypopigmentation located on the upper back.

myositis; and hypomyopathic DM (HDM), with subclinical muscle involvement and skin rash of DM.⁸⁶ DM is well known to be associated with an increased risk of malignancy, and some clinicians consider it a paraneoplastic phenomenon. Approximately 20% of patients with DM are diagnosed with solid organ cancers such as breast, ovarian, genitourinary, lung, and colon within the first 3 to 5 years after the diagnosis of DM is made.⁸⁷

Cutaneous manifestations of DM can precede, parallel, or follow the onset of muscle disease. Amyopathic DM represents greater than 20% of all patients with DM.⁸⁸ A study showed that the risk of malignancy association for amyopathic DM is the same as for classic DM.⁸⁹ However, patients with some forms of amyopathic DM have a higher risk of developing a more severe form of interstitial lung disease that can be rapidly progressive and fatal.⁹⁰ Patients presenting without symptoms of muscle involvement can be easily misdiagnosed, which can lead to a lack of appropriate cancer screening and work-up for pulmonary disease.⁸⁸ Therefore, it is critical to recognize cutaneous features of DM and be able to distinguish this condition from other dermatoses that it can mimic, such as AD, ACD, seborrheic dermatitis, psoriasis, and cutaneous lupus.

The most common and pathognomonic skin lesions of DM are Gottron papules and Gottron sign. Gottron papules are lichenoid, violaceous, flat-topped papules and thin plaques with slight atrophy and subtle scaling overlying the dorsal surface of metacarpophalangeal and interphalangeal joints (Fig. 10).⁸⁴ Gottron sign represents symmetric, slightly scaly, erythematous to violaceous macules and patches overlying extensor tendons of dorsal hands in a linear fashion as well as the dorsal-lateral aspects of finger joints, elbows, knees, and medial malleoli.^{86,87,90} Another hallmark cutaneous feature of DM is a heliotrope rash characterized by symmetric periorbital violaceous erythema that is more pronounced over the upper eyelids.⁹¹

Poikilodermatous skin changes with macular violaceous erythema in photoexposed areas of the posterior shoulders, upper back, and neck are referred to as the shawl sign. When these skin changes are present on the anterior neck and upper chest,



Fig. 10. Gottron papules: lichenoid, violaceous, flat-topped papules coalescing into thin plaques with slight atrophy and subtle scaling overlying the dorsal surface of the metacarpophalangeal and interphalangeal joints.



Fig. 11. V sign of dermatomyositis: poikilodermatous changes with macular violaceous erythema of the anterior neck and upper chest.

they are termed the V sign (**Fig. 11**). The presence of these lesions on the lateral upper thighs and hips is known as the holster sign.⁹⁰

The involvement of the scalp in patients with DM is frequently accompanied by severe pruritus, which can be resistant to systemic treatment and can significantly affect the patient's quality of life.⁹² Erythematous, scaly plaques of the scalp can look like ACD, seborrheic dermatitis, or psoriasis (**Fig. 12**).

Characteristic nailfold changes observed in DM include periungual erythema with telangiectasia of the proximal nail folds, dystrophic and ragged cuticles (Samitz sign), as well as dilated capillary loops with dropout (**Fig. 13**).^{87,91,93}

Mechanic's hands is a known skin finding in DM that presents as hyperkeratosis, scaling, and fissuring of the fingertips, palms, and lateral aspects of the first, second, and third digits.⁹⁴ This cutaneous finding of DM is recognized as a clinical marker of the antisynthetase syndrome, which is associated with arthritis, Raynaud phenomenon, and interstitial pneumonitis.^{93,95} Mechanic's hands of DM can closely mimic atopic, allergic, or irritant contact hand dermatitis and can easily be missed.



Fig. 12. Scalp involvement in patient with dermatomyositis: scaly, violaceous erythema of the superior forehead that extends into the frontal hairline.



Fig. 13. Nailfold changes in dermatomyositis: ragged cuticle of the nailfold. Dilated capillary loops with dropout.

CLINICS CARE POINTS

- Patients with AD are prone to ACD because of their epidermal barrier dysfunction. Therefore, AD and ACD can coexist in this patient population, which can make it even more difficult to differentiate between these two inflammatory skin conditions. Patch testing should be strongly considered in patients with AD in whom dermatitis is worsening, becoming more generalized, or is refractory to topical steroid therapy.³⁶
- When facial psoriasis is suspected, examination of the patient's scalp is important because almost all patients with facial psoriasis also have scalp involvement.^{36,37} Another diagnostic clue may be the presence of fine pinpoint bleeding after gently removing dry scale from the surface of the plaque, called the Auspitz sign.⁴⁰
- Koebnerization phenomenon caused by injury and skin trauma results in worsening of the disease. Scalp psoriasis is vulnerable to koebnerization because of brushing, shampooing, scratching, and mechanical removal of thick scale by many patients who are unaware of this phenomenon.³⁶ History of disease exacerbation caused by trauma can provide a diagnostic clue.
- When considering a diagnosis of inverse psoriasis, asking the patient about genital involvement and performing a genital examination is of paramount importance because the groin is the most affected area in patients with inverse psoriasis, and external genitalia is involved in 80% of patients.^{38,39}
- Checking for nail involvement, which is present in 25% of patients with psoriasis, may provide a diagnostic clue.³⁴ Look for irregular nail pitting, onycholysis, subungual hyperkeratosis, brown oil drop–like spots, nail bed discoloration, and splinter hemorrhages.³⁸

- Most patients have a history of localized plaque psoriasis before the onset of generalized exfoliative dermatitis.⁴¹ Therefore, obtaining personal and family history of psoriasis is crucial to establish a diagnosis. Other clinical clues include presence of palmoplantar keratoderma, nail changes, arthritis, and involvement of scalp, genitoanal, periumbilical, and retroauricular areas.⁹⁷
- Two-feet-one-hand syndrome is a unilateral fungal infection of the hand that occurs with chronic bilateral tinea pedis.⁹⁸ Therefore, when diagnosis of tinea manuum is in question, it is prudent to check the patient's feet for tinea pedis.
- Secondary tinea usually results from autoinoculation of primary tinea pedis.⁵⁰ Check interdigital spaces of the feet for scale, maceration, fissuring, and peeling. Erythema, hyperkeratosis, and scaling of the sole, heel, and sides of the feet in a moccasin distribution can also be present.⁴⁷ Onychomycosis is common in chronic cases. Look for thickened nail plate with yellow discoloration, crumbling, ridging, and subungual debris.⁹⁹
- Patient's history of intense pruritus that is worse at nighttime can provide a clue to the diagnosis of scabies.
- Scabies can occur in adults and children with preexisting eczema. Therefore, it is important to consider the diagnosis of scabies in any patient who presents with widespread dermatitis or pruritus of new onset, or with widespread impetigo.¹⁰⁰
- Hypopigmented MF can closely resemble pityriasis alba, a cutaneous disorder that is common in children with AD. Pityriasis alba is found on the face, neck, and upper trunk, whereas, in hypopigmented MF, face and neck are usually spared.^{70,101}
- Presence of alopecia, erosions, ulcerations, or indurated lesions can be clinical clues to the diagnosis on MF.⁷¹
- Diagnosis of MF palmaris et plantaris should be considered and ruled out in cases of long-standing hand-foot dermatitis that is poorly responsive to treatment or has atypical clinical presentation.⁷¹
- Erythroderma is an uncommon initial presentation of SS; therefore, in cases of nonerythrodermic dermatitis that is refractory to conventional therapy, diagnosis of early SS should be considered. Skin biopsy as well as testing for monoclonal T-cell receptor rearrangement and flow cytometry should be performed.^{102,103}
- SCLÉ lesions can resolve with characteristic vitiligo-like hypopigmentation or telangiectasia that subsides with time and does not lead to atrophy or scarring.^{73,76}
- Heliotrope rash can be easily mistaken for AD or ACD of the eyelids. The important clinical feature that helps to make the correct diagnosis is the presence of periorbital edema, which always accompanies the heliotrope rash. The involvement of the orbicularis oculi muscle is responsible for the periorbital swelling and may result in the sensation of tightness and tenderness of the eyelids.^{87,91}
- Unlike in hand dermatitis, pruritus is not a common feature of mechanic's hands. Also, patients usually deny any history of allergen exposure or the presence of preceding vesicular lesions. Resistance to topical ultrapotent corticosteroid therapy can be another clue to the diagnosis.¹⁰⁴

DISCLOSURE

The authors have nothing to disclose.

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