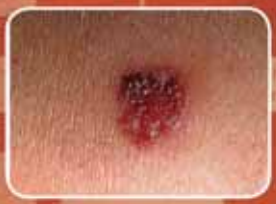

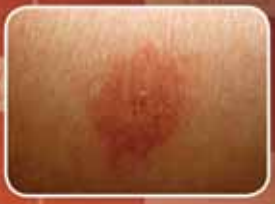

DIFFERENTIAL DIAGNOSIS IN DERMATOLOGY

SECOND EDITION

Klaus F Helm
Galen T Foulke
James G Marks Jr

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KLAUS F HELM MD
Professor of Dermatology and Pathology
Department of Dermatology
Pennsylvania State University College of Medicine
Hershey, Pennsylvania
USA

GALEN T FOULKE MD
Assistant Professor of Dermatology
Department of Dermatology
Pennsylvania State University College of Medicine
Hershey, Pennsylvania
USA

JAMES G MARKS Jr MD
Professor
Department of Dermatology
Pennsylvania State University College of Medicine
Hershey, Pennsylvania
USA

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Preface

While our primary objective in writing *Differential Diagnosis in Dermatology* – enabling healthcare providers to accurately diagnose skin diseases – remains unchanged, we have expanded and enhanced the book in a number of ways.

Firstly, the design of the algorithms and the layout of each chapter have been improved to help the logical flow of the content stand out. We have added short 'Disease discussion' sections at the end of each chapter to provide management guidance on commonly encountered diseases. Lastly, we have included a self-assessment chapter to enable readers to test themselves on the physical signs of cutaneous disease.

Aside from these changes, *Differential Diagnosis in Dermatology 2nd Edition* continues to provide what we hope is a unique and invaluable system of presenting both common and uncommon dermatologic differential diagnoses in a problem-oriented manner. Unlike other text-atlases that catalog diseases in a purely descriptive manner, this book is organized in a way that enables clinicians to work through the diagnostic dilemmas they are likely to encounter in clinical practice.

A templated approach to evaluating skin diseases is the unifying theme. Firstly, diagnostic algorithms at the start of each chapter help the reader to arrive at a shortlist of differential diagnoses. Next, highly structured sets of 'compare and contrast' tables and clinical photographs enable the reader to refine the shortlist still further. The overarching goal is to provide a roadmap that helps the clinician arrive at the correct diagnosis.

While the first edition of *Differential Diagnosis in Dermatology* limited itself to the discussion of differential diagnoses, we have added succinct discussions of the commonest disorders described in the algorithms and tables. These cover etiology, clinical features and recommended treatment. Combined with the unique approach to working through the differential diagnosis of skin lesions, we hope the inclusion of this management information enhances the book's value to primary care providers and dermatologists by helping them diagnose and treat the many patients they see with cutaneous skin disorders.

Klaus F Helm
Galen T Foulke
James G Marks Jr
October 2017

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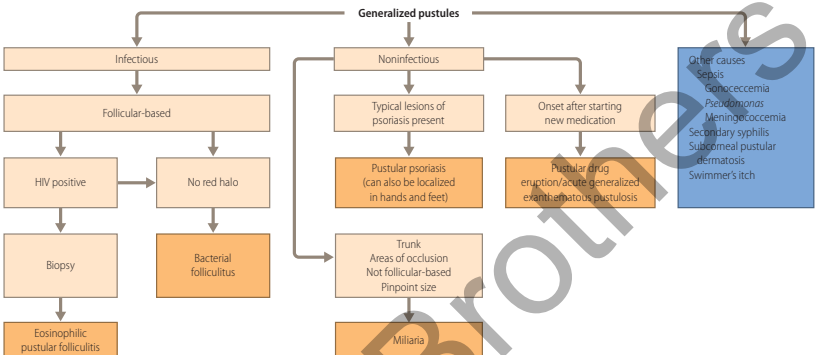
Chapter 12 Generalized pustular eruptions



INTRODUCTION

A pustule is a circumscribed 'blister' of the skin containing pus. Microscopic evaluation reveals a focal collection of neutrophils or eosinophils. Pustular eruptions can be subdivided into either localized or generalized eruptions. Pustules can also be categorized according to cause (infectious versus noninfectious). The most common localized noninfectious form of pustular eruptions is acne, which is discussed in Chapter 2. Causes of localized infectious pustules such as impetigo and candidiasis are also discussed in Chapters 2 and 3.

Generalized non-infectious pustular eruptions include pustular psoriasis, acute generalized exanthematous pustulosis, and miliaria. The diagnosis is usually relatively straightforward, since patients with pustular psoriasis most often have classic psoriatic lesions elsewhere. Acute generalized exanthematous pustulosis start after the onset of administration of a drug, and miliaria occurs in occluded areas in patients who have been sweating. Generalized infectious pustular eruptions include eosinophilic pustular folliculitis (Ofuji's disease), which is likely due to an as-yet-unidentified organism, and bacterial folliculitis.

1. EOSINOPHILIC PUSTULAR FOLLICULITIS VERSUS BACTERIAL FOLLICULITIS

Features in common: follicular papules and pustules

Figure 12.1.1 Bacterial folliculitis.

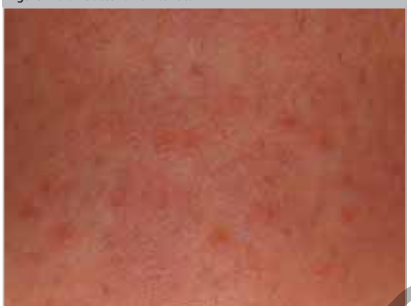


Figure 12.1.2 Eosinophilic pustular folliculitis.

Distinguishing features

	Bacterial folliculitis	Eosinophilic pustular folliculitis
Physical examination		
Morphology	No surrounding halo (except in pseudomonal folliculitis, also known as hot-tub folliculitis) No urticarial papules	Red halo surrounding pustules Urticarial papules
Distribution	Trunk, buttocks, and thighs Face rarely involved	Trunk and proximal extremities favored Face may be involved
History		
Symptoms	Pruritus	Pruritus
Exacerbating factors	Diabetes	None known
Associated findings	None	None
Epidemiology	No age or sex predilection Most commonly seen in normal population but can also occur in patients with human immunodeficiency virus disease	Most common in patients with acquired immunodeficiency syndrome or individuals of Japanese heritage Rarely seen in normal population
Biopsy	No Pustule filled with neutrophils connected to hair follicle	Yes Pustule filled with eosinophils connected to hair follicle
Laboratory	Culture positive for *Staphylococcus aureus*	Culture negative
Outcome	Good	Chronic

Differential diagnosis of follicular based papules and pustules

- Acne vulgaris
- Acneiform drug eruption
- Bacterial folliculitis (*Staphylococcus, Pseudomonas*)
- Eosinophilic pustular folliculitis
- Folliculitis decalvans
- Fox Fordyce disease
- Herpes simplex/zoster
- Hidradenitis suppurativa
- Insect bites
- Keratosis pilaris
- Lichen planus/lichen planopilaris
- Lupus miliaria disseminatum faciei
- Majocchi's granuloma
- Miliaria (should be eccrine based)
- Newborn
  - Candidiasis
  - Erythema toxicum neonatorum
  - Pustular melanosis of infancy
- Pyoderma gangrenosa
- Scurvy
- Sycosis barbae

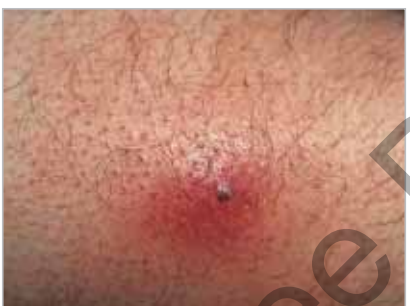


Figure 12.1.3 Bacterial folliculitis. *Clue to diagnosis:* furuncle (follicular based absscess).

Figure 12.1.4 Bacterial/*Pseudomonas* folliculitis. *Clue to diagnosis:* hot tub use.

2. MILIARIA RUBRA VERSUS BACTERIAL FOLLICULITIS

Features in common: generalized papules and pustules

Figure 12.2.1 Bacterial folliculitis.

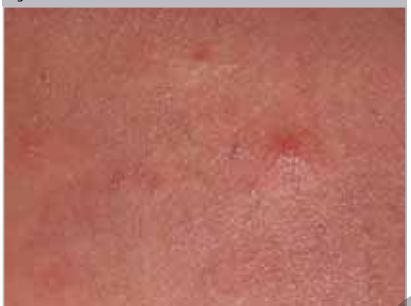


Figure 12.2.2 Milaria.

Distinguishing features

	Bacterial folliculitis	Miliaria rubra
Physical examination		
Morphology	Primary lesion: pustule, no vesicles Excoriations common	Primary lesion: small papules and vesicles (pustules secondary finding) Excoriations rare
Distribution	Trunk, buttocks, and thighs	Trunk or in areas of occlusion like diaper area in children
History		
Symptoms	Pruritus	No pruritus
Exacerbating factors	Use of hot tub in *Pseudomonas* folliculitis None	No hot tub use Occlusive clothing Hot humid climate
Associated findings	Diabetes	Fever, profuse sweating
Epidemiology	No age or sex predilection	Most common in babies and infants
Biopsy	No Pustule filled with neutrophils connected to hair follicle	No Dilated and obstructed eccrine duct
Laboratory	Culture positive for *Staphylococcus aureus*	Culture negative
Outcome	Good	Good

Differential diagnosis of generalized pustular eruptions

- Common causes
  - Folliculitis
  - Impetigo
  - Miliaria
  - Pustular drug eruption/acute generalized exanthematous pustulosis
  - Pustular psoriasis
  - Transient neonatal pustular melanosis
- Rarer causes
  - Ecthyma gangrenosum
  - Eosinophilic pustular folliculitis (Ofuji's disease)
  - Erythema toxicum neonatorum
  - Gonococcemia
  - IgA pemphigus
  - Impetigo herpetiformis
  - Pyoderma gangrenosum
  - Reiter's disease
  - Secondary syphilis
  - Subcorneal pustular dermatosis
  - Swimmer's itch

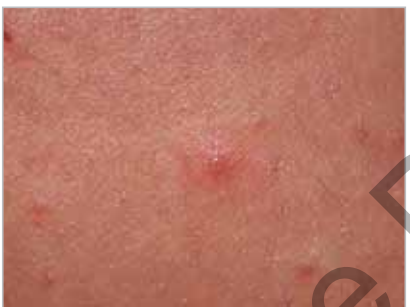


Figure 12.2.3 Bacterial folliculitis. *Clue to diagnosis:* follicular based pustule.

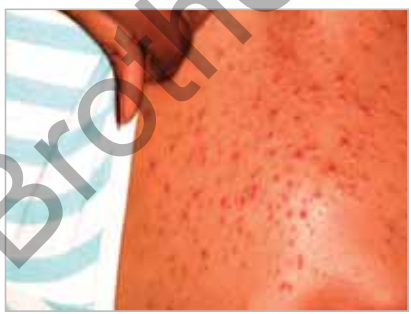


Figure 12.2.4 Miliaria rubra: red bumps localized to eccrine orifices.

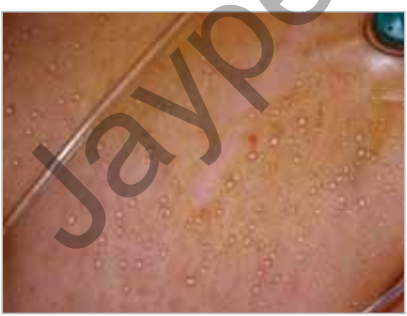


Figure 12.2.5 Miliaria pustulosa: pustules localized to eccrine orifices.

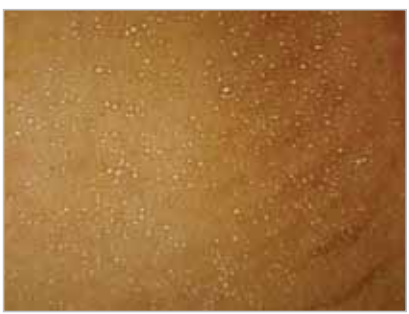


Figure 12.2.6 Miliaria crystalline: clear fluid filled papules.

3. PUSTULAR PSORIASIS VERSUS ACUTE GENERALIZED EXANTHEMATOUS PUSTULOSIS

Features in common: generalized pustules

Figure 12.3.1 Pustular psoriasis.

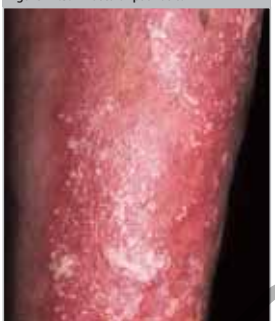
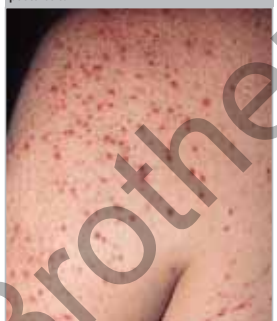


Figure 12.3.2 Acute generalized exanthematous pustulosis.



Distinguishing features

	Pustular psoriasis	Acute generalized exanthematous pustulosis
Physical examination		
Morphology	Red papules and plaques Silvery-colored scale Pustules not follicular based Annular plaques/patches with peripheral pustules may be present	No plaques No scale Pustules may be follicular based No annular lesions
Distribution	Scalp and predilection for extensor surfaces of body	Truncal, no predilection for extensor surfaces
History		
Symptoms	No new drug Fever	New drug Fever rare
Exacerbating factors	Tapering of oral steroids, infections, pregnancy, medications, irritating topical medications, sunburn	None
Associated findings	Positive family history in 10–20% of patients Improves with sun exposure Arthritis in some patients Onychodystrophy	No family history of psoriasis No relationship to sun exposure No arthritis No onychodystrophy
Epidemiology	Any age, no sex predilection	Usually adults
Biopsy	Yes Psoriasiform epidermal hyperplasia with neutrophil microabscesses in epidermis and stratum corneum	Sometimes Subcorneal pustule Occasionally vasculitis Eosinophils usually present
Laboratory	Increased erythrocyte sedimentation rate (ESR) Leukocytosis Hypocalcemia	Slight leukocytosis
Outcome	Chronic disease	Resolves after drug is discontinued

Common drugs causing acute generalized exanthematous pustulosis

- Antibiotics
- Calcium channel blockers
- Carbamazepine
- Hydroxychloroquine
- Sulphonamides
- Terbinafine

DISEASE DISCUSSION

Acneiform drug eruptions

Definition and etiology

A variety of medications can produce an acneiform eruption resembling acne (see next).

Drugs causing acneiform eruption

- Androgens
- Anti-seizure medications
- Corticosteroids
- Cyclosporine
- Epidermal growth factor inhibitors
- Isoniazid
- Lithium
- Halogens (iodide, bromide)
- Sirolimus
- Tricyclic antidepressants
- Tyrosine kinase inhibitors
- Vitamin B_{12}

Clinical features

Acneiform pustular drug eruptions resemble acne except that lesions are predominantly monomorphous pustules and comedones are rarely found. Acneiform drug eruptions are most commonly produced by androgens, oral corticosteroids, isoniazid, lithium, halogens, and epidermal growth factor inhibitors. Onset of acneiform eruption in an elderly person or acne unresponsive to therapy should raise the suspicion of possible drug being the culprit.

Treatment (Table 12.1)

Since the medications causing the acneiform rash often need to be continued, therapy is similar to treatment of acne vulgaris.

Table 12.1 Treatment acneiform drug eruptions

Treatment	Precautions
Universal	
Stop or change the medication if possible	No always possible in some patients if medication is medically necessary such as in EGF inhibitors for cancer treatment
Mild-to-moderate disease	Treat as regular acne
Mild disease	
Tretinoin cream (0.025, 0.05, 0.1%) Tretinoin gel (0.01, 0.025%) Adapalene cream (0.1% Adapalene gel (0.1, 0.3%)	May be too irritating for elderly patients Apply on dry skin and small amount or may be too irritation Apply at nighttime since light may inactivate Do not pimple dab since trying to prevent new lesions
Moderate disease	
Use one of above and add benzyl peroxide and/or topical antibiotics Clindamycin Erythromycin	May be irritating Rare allergic reactions
Severe disease	
Add oral antibiotics	
Minocycline Doxycycline	Photosensitivity, Can produce blue discoloration of gums and skin Rare lupus like syndrome with fevers, malaise, rash Photosensitivity Esophagitis Vaginal yeast infections
Other considerations for women	
Oral contraception	Risk of stroke, thrombus especially in smokers and older women
Spironolactone 50–100 mg q day	Avoid pregnancy
Miscellaneous treatments	
Isotretinoin	Class X: causes birth defects Rare cases of depression and suicide Rare cases of coexistent or worsening of inflammatory bowel disease
Azelaic acid 20% cream	Only treatment that is class B and can safely be used during pregnancy
Dapsone 5% gel	Alternative topical if allergic to others

Bacterial folliculitis

Definition and etiology

Bacterial folliculitis is an infection of hair follicles, most often due to *Staphylococcus aureus* but also sometimes due to *Pseudomonas aeruginosa*. *Pseudomonas* folliculitis is most commonly seen in patients who have been using hot tubs.

Bacterial folliculitis can occur on anybody site that has hair follicles. Folliculitis decalvans and dissecting cellulitis are specific variants that occur on the scalp. Sycosis barbae is a form that occurs on the beard area. The most common sites of bacterial folliculitis are the legs, thighs, and trunk. Examination reveals follicular-based papules and pustules that often have a central hair. The lesions are highly pruritic

and often excoriated. Because of chronic scratching, eczematous changes may be present.

The differential diagnosis includes miliaria pustulosa, acne, dermatitis herpetiformis, *Pityrosporum* folliculitis, and Grover's disease (transient acantholytic dermatosis). Unlike in acne, comedones are not present in folliculitis, and the distribution of lesions is usually different. Miliaria pustulosa is caused by occlusion of eccrine glands. Miliaria most commonly occurs suddenly after an episode of severe sweating in sites of occlusion, such as the trunk. The lesions are usually smaller than lesions of folliculitis, and small vesicles and pustules are present. Grover's disease is a very pruritic, nondescript papular truncal eruption (pustules are usually absent) that is predominantly found in adult men. *Pityrosporum* folliculitis is morphologically identical to bacterial folliculitis but is due to *Pityrosporum orbiculare*, a commensal yeast. It occurs more commonly in immunosuppressed individuals and may uncommonly be a presenting sign of underlying disease, such as Hodgkin's disease. The diagnosis is made clinically, and positive culture results are confirmatory. In most cases of folliculitis, a biopsy is not necessary.

Treatment (Table 12.2)

Occlusive clothing should be avoided. In severe cases, oral antibiotics effective against *Staphylococcus aureus* should be used. In milder cases, topical antibiotics and antibiotic soaps are effective. In some cases, recurrence is common and the disease difficult to eradicate despite use of appropriate antibiotics and antibacterial soaps. Work-up for immune deficiency or diabetes, however, is usually still negative

Eosinophilic pustular folliculitis

Definition and etiology

Eosinophilic pustular folliculitis (Ofuji's disease) is an idiopathic disorder hypothesized to be infectious. This form of folliculitis is characterized by an eosinophilic inflammatory infiltrate within and surrounding hair follicles.

Clinical features

Three forms of Ofuji's disease exist. The classical form was originally described as a form of pruritic folliculitis of the scalp in Japanese men. More commonly in the United States is the immunosuppressed form most frequently occurring in individuals with human immunodeficiency virus infection. Finally, there also is a form occurring in children. In all types, follicular papules and pustules are seen. Unlike in bacterial folliculitis, urticarial papules are also present, and the pustules are frequently surrounded by a bright red urticarial halo. In the classical form, annular papules and pustules occur on the scalp and trunk. Although the diagnosis can be suspected in patients with acquired immunodeficiency syndrome, diagnosis ultimately depends on the histologic finding of an eosinophilic pustule within the follicular infundibulum. In the infantile form annular lesions are usually absent and there is a predilection for the scalp.

The differential diagnosis includes Grover's disease (transient acantholytic dermatosis) and miliaria pustulosa. Grover's disease primarily affects adult men and is characterized by pruritic nondescript papules that are not follicular based. A characteristic feature of Grover's disease is severe pruritus with minimal clinical findings. In miliaria, the lesions are smaller and not follicular based. They appear suddenly after severe sweating.

Treatment (Table 12.3)

Treatment is difficult. Topical or systemic steroids, Indomethacin, ultraviolet light therapy, itraconazole, topical permethrin, and oral metronidazole have been reported to help. HIV related cases improve with strong antiviral therapy.

Miliaria

Definition and etiology

Miliaria is an eruption due to obstruction of eccrine glands. It can be subdivided into miliaria crystallina, rubra, or profunda depending on the site of obstruction. The sites of obstruction are the stratum corneum in miliaria crystallina, the epidermis in miliaria rubra, and within the dermis in miliaria profunda.

Table 12.2 Treatment of bacterial folliculitis

Treatment	Precautions
Universal	
Consider culture to confirm diagnosis and determine sensitivities	
Treat any source of *Staphylococcus*	Pets can occasionally carry *Staphylococcus*
If chronic consider culture nasal nares	Nasal carriage often requires cocktail of antibiotics to clear
Avoid tight fitting clothes	
Women who shave legs with the grain	Frequent changing of razor blades may be helpful
Benzyl peroxide or Hibiclens wash	May be irritating
Severe cases	
Oral antibiotics as per culture	

Table 12.3 Treatment of eosinophilic pustular folliculitis

Treatment	Precautions
Universal	
Skin biopsy or scraping to confirm the diagnosis	
Oral antihistamines	May help with pruritus
Mild disease	
Topical corticosteroid bid	Atrophy, steroid-induced acne
Topical calcineurin inhibitor bid	May burn or sting
Permethrin 5% cream q day	Irritation
Moderate-to-severe disease	
Indomethacin 50– 75mg q day	May be treatment of choice for classical variant Gastrointestinal upset common Risk of bleeding
UVB therapy	Risk of sunburn and skin cancer
Short course oral corticosteroids 1 mg/kg/day	Steroid side effects: hyperglycemia, insomnia, hypertension, fluid retention
Itraconazole 100–400 mg/day	Liver toxicity
Metronidazole 250 mg tid	Has Antabuse affect when taken with alcohol May cause nausea, vomiting, metallic taste
Antiviral therapy	HIV associated cases may improve

Clinical features

Miliaria presents as an acute pruritic eruption after an episode of severe sweating. Covered sites such as the trunk or diaper are most commonly involved. In miliaria crystallina, small pinpoint-sized vesicles are seen. In miliaria rubra, small vesicles, papules, and pustules are present. In miliaria profunda, papular lesions predominate. The major differential diagnostic considerations are folliculitis and Grover's disease. The lesions of folliculitis are predominantly pustular and not vesicular, as in miliaria. Folliculitis is centered on hair follicles. Although sweating may have a pathogenic role in producing the lesions of Grover's disease, it is typically a chronic nonpruritic eruption in elderly men that requires a biopsy for definitive diagnosis.

Treatment (Table 12.4)

The eruption of miliaria is usually self-limited and does not require therapy. Wearing occlusive clothing and sweating should be minimized. Cool baths, compresses, and air conditioning may be helpful.

Table 12.4 Treatment of miliaria rubra

Treatment
Avoid sweating
Avoid occlusive clothing
Cool baths
Compresses
Topical corticosteroids if pruritic

Acute generalized exanthematous pustulosis

Definition and etiology

Acute generalized exanthematous pustulosis is a severe exanthematous and pustular skin rash usually associated with high fevers, which is usually due to a variety of different medications.

Clinical features

Acute generalized exanthematous pustulosis is most commonly produced by a variety of different antibiotics and can be confused with pustular psoriasis or an exanthem if the pustules are obscured by the exanthematous portion of the rash. Occasionally a variety of infections have also been reported to be etiologic agent. The lesions are predominantly truncal; numerous pustules on an erythematous base are found; and the pustules are not follicular centered. As in pustular psoriasis, fever is frequently present. The onset of the rash 1–11 days after ingestion of a medication, the lack of a family history of psoriasis, and the lack of classic psoriatic lesions help distinguish pustular drug eruptions from pustular psoriasis. The differential could also include DRESS (drug reaction with eosinophilia and systemic symptoms). Patients with DRESS are very sick, have lymphadenopathy, evidence for hepatitis, and peripheral eosinophilia.

Treatment (Table 12.5)

Acute generalized exanthematous pustulosis may improve spontaneously after the medication is discontinued. Supportive skin care is frequently all that is needed, but some patients have been treated with oral prednisone.

Table 12.5 Treatment of acute generalized exanthematous pustulosis

Treatment	Precautions
Universal	
Eliminate causative medication	Eruption is often self-limited and resolves in approximately 2 weeks, and no treatment may be necessary after medication eliminated
Consider biopsy to exclude pustular psoriasis	
Mild disease	
Topical corticosteroids such as triamcinolone 0.1% cream bid	May produce atrophy and steroid acne Avoid in intertriginous areas and on face
Moderate-to-severe disease	
Oral corticosteroids	Steroid side effects: hyperglycemia, insomnia, hypertension, fluid retention

Psoriasis

Definition and etiology

Psoriasis is an inflammatory disease characterized by increased epidermal proliferation. The cause of psoriasis is unknown, but abnormal epidermal kinetics, activation of the immune system within the skin, and genetic factors must be taken into account.

Clinical features

Approximately one-third of individuals with psoriasis have a positive family history. Psoriasis is a relatively common skin disease, affecting about 2% of the population of the United States. Asians are less commonly affected. The most common age of onset is during the third decade, but psoriasis can present at any age. In most cases of psoriasis, examination reveals bright red erythematous plaques covered with white to silvery-colored scale. Areas of predilection include the elbows, knees, scalp, and sacrum. Extensor surface involvement predominates. Pitting of the nails and onychodystrophy occur frequently. Nails may appear to have an oil drop under them and small pits. Pustular psoriasis is a rare variant of psoriasis in which pustules are found. Pustular psoriasis can be further divided into four subtypes: palmoplantar acral pustular psoriasis (see Chapter 4), acute generalized pustular psoriasis of von Zumbusch, subacute annular psoriasis, and a mixed type. Recently, it has been discovered that some patients with pustular psoriasis have an Il-36 receptor antagonist deficiency or mutation.

Acute generalized pustular psoriasis of the von Zumbusch type occurs in patients with a history of classic psoriasis. The generalized pustular flare-up is precipitated by a variety of factors, such as corticosteroid withdrawal, pregnancy, infection, sunlight, or irritating topical therapy (e.g., coal tar or anthralin). Patients are seriously ill with fever and have an elevated erythrocyte sedimentation rate as well as leukocytosis and arthritis. Pre-existing lesions become bright red and develop pustules. Sheets of pustules spread to previously uninvolved skin to cover the entire integument. Isolated pustules, annular lesions, and plaques of pustules are found. In the subacute annular form of pustular psoriasis, the lesions start as annular areas of erythema that become raised and edematous and have a serpiginous appearance. Eventually, pustules appear at the advancing edge of the lesions. Unlike in the acute form, lesions develop slowly, and patients are not as sick. In the mixed form, features of both the acute and the subacute annular form are seen.

The major differential diagnostic considerations include acute generalized exanthematous pustulosis, subcorneal pustular dermatosis (Sneddon-Wilkinson, disease), IgA pemphigus, bacterial folliculitis, and acrodermatitis continua (Hallopeau's acrodermatitis). Acute generalized exanthematous pustulosis occurs 3 to 5 days after onset of administration of certain medications. Patients are usually not as sick as patients with pustular psoriasis, do not have high fever, have minimal leukocytosis, and do not have a history of preceding psoriatic lesions.

In subcorneal pustular dermatosis, the lesions are mostly localized to flexural areas such as the axilla and groin. A characteristic finding is pustules with a meniscus of clear overlying fluid. In acrodermatitis continua, which may also be a variant of pustular psoriasis, the pustules predominantly start around fingernails and cause confusion with paronychia infection.

Treatment (Table 12.6)

The treatment of choice for generalized pustular psoriasis is oral acitretin, cyclosporine, methotrexate, or infliximab. Alternative therapies include standard therapies for psoriasis such as TNF inhibitors, Il-17 inhibitors, Il-12, 23 inhibitors, apremilast, phototherapy, etc.

Table 12.6 Treatment of psoriasis

Universal	Precautions
Look and eliminate trigger factors such as streptoccocal infections and medications	
Mild-to-moderate disease	
Topical steroids bid	Atrophy, steroid induced acne
Topical calcipotriene bid	Slight irritation Overuse could cause hypercalcemia
Topical tar preparations such as liquor carbonis detergens 5% bid	Irritation
Topical tazarotene 0.05%, 0.1% cream or gel	Pregnancy category X Frequently irritating
Anthralin	Stains clothing, skin and irritation
Severe disease	
Acitretin 25–75 mg daily	Dry skin, hair loss, elevated serum lipids, birth defects Periodic liver function tests, and lipid profile Birth defects
Methotrexate 10–25 mg q week	Risk of liver toxicity, anemia, nausea, vomiting
Cyclosporine 5 mg/kg/day	Hypertension, renal insufficiency, immunosupression
TNF inhibitors Etanercept: start 50 mg SC 2×/week × 3 months than 50 mg SC q week Adalimumab: start 80 mg SC on day one then 40 mg sq day eight followed by 40 mg SC q 2 weeks Infliximab: 5 mg/kg IV week 0, 2 and 6 then q8 weeks	Risk of severe infection and increased risk of malignancies especially lymphoma
IL-12, 23 blocker Ustekinumab (weight dependent dosage) for less than 100 kg: 45 mg SC × 1 on week 0 and week 4 then q12 weeks	No black box warning, but still risk of severe infections and possibly malignancy
IL-17 blockers Secukinumab: start 300mg SC q week × 5 than 300 mg SC q4 weeks ixekizumab: start 160 mg SC × 1 then 80 mg SC q 2 weeks × 12 week. Then once a month Brodalumab: start 210 mg SC at weeks 0, 1 and 2, then 210 mg SC q 2 weeks	Black box warning only for brodalumab for suicidal ideation and risk Risk of severe infection especially candidal Risk of colitis
Ultraviolet light-narrow band UvB	Risk of burning and skin cancer
PUVA (psoralen plus UVA light)	Risk of burning and increased risk of skin cancer
Apremilast 30 mg orally bid	Risk of depression, diarrhea, nausea, headache, weight loss