PPID | Private Practice Infectious Disease

MIDC METRO INFECTIOUS DISEASE CONSULTANTS

Five Case-Based Vignettes in Diagnostic Dermatology for Infectious Disease Specialists

Russell Petrak, MD [1,*]

[1] Metro Infectious Disease Consultants, Burr Ridge, IL 60527, USA
* Corresponding author: rpetrak@midcusa.com

Submitted: 19 October 2026, accepted: 19 October 2026, published: 31 December 2026

Abstract: Cutaneous findings are frequently the presenting sign of systemic disease, and recognizing the pattern, distribution, and evolution of a rash can shorten the path from bedside observation to unifying diagnosis. This review summarizes 5 clinically distinct entities: keratoderma blennorrhagica, a psoriasiform palmoplantar eruption of reactive arthritis; erythema marginatum, the evanescent annular rash of acute rheumatic fever; Sweet's syndrome, an acute febrile neutrophilic dermatosis, which is a reaction pattern linked to infection, malignancy, and drug exposure; pyoderma gangrenosum, an ulcerative neutrophilic dermatosis in which pathergy is a recognized feature; and fixed drug eruption, a T-cell-mediated reaction that recurs at a fixed cutaneous site with re-exposure to a culprit drug. For each condition, we outline the clinical vignette, pathophysiology, morphology, associated systemic disease, differential diagnosis, and management. We conclude with a comparative summary and key diagnostic pearls that link these otherwise unrelated dermatoses.

Keywords: skin manifestations; neutrophilic dermatoses; reactive arthritis; drug eruptions

How to cite: Petrak, R. Five Case-Based Vignettes in Diagnostic Dermatology for Infectious Disease Specialists. *Priv. Pract. Infect. Dis.*, 2026, 6(4): 24; doi:10.55636/ppid06040024.

© 2026 Copyright by Author. Licensed as an open access article using a CC BY 4.0 license.

Introduction

The skin is an accessible "window" to internal disease. Knowing a rash's morphology, tempo, and distribution narrows the differential before a single laboratory test is drawn. This review considers 5 conditions that share a teaching format—a brief clinical vignette followed by a diagnostic "clue." The conditions differ markedly in mechanism: 2 are cutaneous signs of classic post-infectious immune-mediated syndromes (keratoderma blennorrhagica, erythema marginatum), 2 are neutrophilic dermatoses that sit on a shared pathophysiologic spectrum (Sweet's syndrome,

pyoderma gangrenosum), and one is a delayed-type drug hypersensitivity reaction with a unique site-specific memory (fixed drug eruption). Presenting them together highlights a broader principle: neutrophil-rich inflammation can manifest as a pustular keratoderma, a tender plaque, or a deep ulcer depending on the trigger and the tissue involved, while superficially similar "rings" or "target" lesions can represent entirely different diseases (erythema marginatum versus erythema multiforme) with divergent workups and prognoses.

1. Keratoderma Blennorrhagica

> ***Illustrative case:*** *A 35-year-old sexually active male presents with hyperkeratotic, crusted lesions on the palms and soles and a painless penile lesion.*

Overview

Keratoderma blennorrhagica (KB) is the characteristic cutaneous manifestation of reactive arthritis, an immune-mediated arthritis that follows a genitourinary or gastrointestinal infection by days to weeks. It occurs predominantly in young adult males and is strongly associated with the human leukocyte antigen B27 (HLA-B27) allele, reflecting a shared post-infectious, immune-mediated mechanism with the joint and mucosal disease of reactive arthritis rather than a primary infection of the skin itself [1,2].

Etiology and Triggers

The most common antecedent organism is *Chlamydia trachomatis*, acquired sexually. A gastrointestinal trigger is also well-described, with *Salmonella*, *Shigella*, *Yersinia*, and *Campylobacter* species implicated following an episode of infectious enteritis [2].

Clinical Presentation (Figure 1)

- Hyperkeratotic, crusted, pustular lesions with a psoriasiform appearance (Figure 1A,B)
- Palms and soles are the most characteristic sites
- Circinate balanitis, a painless, serpiginous penile lesion, frequently accompanies the skin findings

Associated systemic features

KB occurs in the context of reactive arthritis, and clinicians should assess for the classic triad of asymmetric oligoarthritis, urethritis or cervicitis, and conjunctivitis or uveitis, as well as other mucocutaneous lesions such as circinate balanitis [1].

Histopathology

Biopsy shows psoriasiform epidermal hyperplasia, parakeratosis, and neutrophilic pustules—a pattern that is histologically indistinguishable from pustular psoriasis, underscoring that the diagnosis of KB is clinical and contextual rather than purely histologic [1].

Differential Diagnosis

- Pustular psoriasis
- Palmoplantar keratoderma (other causes)
- Secondary syphilis
- Tinea manuum/pedis



(**A**) (**B**)

Figure 1: Keratoderma Blennorrhagica. (**A**) Hyperkeratotic, crusted, psoriasiform plaques on the plantar surfaces [3]. (**B**) Classic keratoderma blennorrhagica of the sole [4]. VA.gov | Veterans Affairs. Accessed November 10, 2026. https://www.hiv.va.gov/provider/image-library/dermatological.asp?post=1&slide=6.

Management and Prognosis

Treatment targets the underlying infection alongside the cutaneous disease [2]. Topical corticosteroids and keratolytics such as urea or salicylic acid are used for the skin lesions, with systemic therapy reserved for severe disease. Skin findings generally improve as the reactive arthritis is controlled, although recurrence is possible and overall prognosis tracks the course of the systemic disease.

2. Erythema Marginatum

> ***Illustrative case:*** *A 27-year-old with congestive heart failure develops a painless, evanescent rash on the trunk and arms—sometimes called the "intern's rash" because it can appear and disappear within hours of examination.*

Overview

Erythema marginatum is an evanescent annular or serpiginous eruption that represents a major Jones criterion for acute rheumatic fever. Its hallmark is transience: Individual lesions can appear and resolve over a matter of hours, so the rash may be missed entirely [5].

Clinical Presentation (Figure 2)

- Annular or serpiginous erythematous rings with central clearing and raised borders (Figure 2A,B)
- Non-pruritic and painless
- Favors the trunk and proximal limbs, characteristically spares the face
- Associated with acute rheumatic fever, which follows group A streptococcal pharyngitis



Figure 2: Erythema marginatum. (**A**) Faint, serpiginous erythematous rings on the trunk [6]. (**B**) Annular lesions with raised erythematous borders and central clearing [7]. Rheumatic fever, erythema marginatum. DermNet®. October 26, 2023. Accessed September 10, 2026. https://dermnetnz.org/topics/rheumatic-fever.

Differential Diagnosis: Erythema Multiforme (Figure 3)

The principal mimicker is erythema multiforme (EM), and distinguishing between erythema multiforme and erythema marginatum has direct implications for workup and management.

Erythema multiforme

- Target ("bull's-eye") lesions with 3 discrete zones (Figure 3A)
- May burn or itch; mucosal erosions are painful, whereas erythema marginatum is asymptomatic
- Fixed lesions that persist for days rather than hours
- Favors acral and extensor surfaces and may involve mucous membranes (Figure 3B)
- Triggered by infection (particularly herpes simplex virus [HSV] and *Mycoplasma pneumoniae*) or by medications



Figure 3: Erythema Multiforme. (**A**) Target lesions on an extremity [8]. Erythema multiforme image. DermNet®. December 5, 2023. Accessed September 10, 2026. https://dermnetnz.org/imagedetail/8270-erythema-multiforme. (**B**) Acral target lesions on the palms and hands [9]. Cutaneous adverse reaction to anticonvulsant, erythema multiforme target lesions image. DermNet®. December 5, 2023. Accessed September 10, 2026. https://dermnetnz.org/imagedetail/8133-cutaneous-adverse-reaction-to-anticonvulsant-erythema-multiforme-target-lesions.

Comparison Chart

Feature	Erythema Marginatum	Erythema Multiforme
Lesion type	Annular/serpiginous rings, central clearing	Target (bull's-eye) lesions
Symptoms	Asymptomatic	Pruritic or painful
Duration	Evanescent (hours)	Fixed (days)
Distribution	Trunk, proximal limbs	Hands, feet, extensor surfaces
Face	Spared	May be involved
Mucosa	Absent	Common in erythema multiforme major
Cause	Post-streptococcal immune reaction	Infection (eg, HSV), drugs
Systemic link	Acute rheumatic fever	Hypersensitivity reaction
Key exam clue	Jones major criterion	Target lesions $\pm$ mucositis

Broader Differential

Two additional "ring" mimics deserve mention. Lyme disease produces erythema migrans, an expanding annular lesion with a very different tempo (days rather than hours) and a clear exposure history. Urticarial drug eruptions can also present with annular, evanescent wheals and a temporal relationship to a new medication. Associated pruritus favors a drug etiology over erythema marginatum.

Clinical Significance

Because erythema marginatum is one of the major Jones criteria, its recognition should prompt an evaluation for acute rheumatic fever, including assessment for carditis, arthritis, chorea, and subcutaneous nodules, together with confirmation of a preceding group A streptococcal infection [5].

3. Sweet's Syndrome (Acute Febrile Neutrophilic Dermatosis)

> ***Illustrative case:*** *A 48-year-old male presents with new-onset fever, a tender rash, and pancytopenia; further workup reveals a diagnosis of acute myeloid leukemia (AML).*

Definition and Etiology

Sweet's syndrome is an acute neutrophilic dermatosis characterized by fever and tender, erythematous plaques or nodules. It is a reactive process rather than an infection, and it presents in 3 clinical settings: classic (idiopathic) disease, which often follows an upper respiratory or gastrointestinal infection; malignancy-associated; and drug-induced disease [10,11].

Diagnostic Approach

The diagnosis rests on the combination of abrupt onset, fever, an identifiable trigger from the categories above, response to systemic corticosteroids, and laboratory evidence of inflammation—an

elevated erythrocyte sedimentation rate, leukocytosis, and elevated C-reactive protein [11]. In malignancy-associated disease, neutrophilia may be absent.

Classic Clinical Appearance (Figure 4)

- Tender, erythematous papules and plaques
- Common sites include the face, neck, and upper extremities



Figure 4: Sweet's syndrome. Tender, erythematous, edematous papules and plaques on (**A**) the posterior neck Acute febrile neutrophilic dermatosis image. DermNet®. November 10, 2024. Accessed September 10, 2026. https://dermnetnz.org/imagedetail/24265-acute-febrile-neutrophilic-dermatosis. (**B**) the face and neck [12]. Sweet syndrome image. DermNet®. December 5, 2023. Accessed September 10, 2026. https://dermnetnz.org/imagedetail/8426-sweet-syndrome.

Associated conditions (classic/idiopathic Sweet's)

- Upper respiratory or gastrointestinal infections
- Inflammatory bowel disease
- Pregnancy
- Autoimmune disease

Malignancy-Associated Sweet's Syndrome

In a pooled analysis, approximately 21% of patients had an associated malignancy. The eruption may precede, follow, or arise concurrently with an underlying malignancy, and hematologic malignancies are more commonly implicated than solid tumors. Among hematologic malignancies, AML accounts for approximately 42% of associated cases, making Sweet's syndrome an important cutaneous clue to an occult or newly diagnosed leukemia—as illustrated in the illustrative case above [10,11].

Drug-Induced Sweet's Syndrome

Recognized culprits include granulocyte colony-stimulating factors (G-CSF), antibiotics such as trimethoprim–sulfamethoxazole, and immune checkpoint inhibitors. Drug-induced disease typically develops approximately 2 weeks after exposure to the offending agent [10].

Differential Diagnosis

- Erythema multiforme
- Cellulitis
- Pyoderma gangrenosum
- Vasculitis

Treatment and Prognosis

Systemic corticosteroids are first-line therapy, with potassium iodide and colchicine as first-line alternatives and indomethacin, dapsone, or cyclosporine as second-line options [10]. Prognosis is favorable with treatment, provided any underlying cause (infectious, malignant, or drug-related) is identified and managed concurrently.

4. Pyoderma Gangrenosum (Figure 5)

> ***Illustrative case:*** *A 45-year-old woman with a chronic, non-healing ulcer is found to have underlying Crohn's disease.*

Definition and Pathophysiology

Pyoderma gangrenosum (PG) is a rare inflammatory neutrophilic dermatosis characterized by progressive, painful cutaneous ulceration. Despite its name, it is non-infectious. It is driven by a dysregulated innate immune response, with neutrophil dysfunction, cytokine dysregulation, and genetic predisposition all implicated. Biopsy characteristically shows a sterile, dense neutrophilic infiltrate [13].

Pathergy is a cardinal and clinically important feature: Minor trauma or surgical manipulation provokes an exaggerated wound response, and lesions characteristically worsen with debridement. Between 50% and 70% of patients have an underlying systemic disease, although the activity of the skin disease does not always parallel the course of that systemic condition [13,14].

Clinical Variants

Variant	Frequency	Key Features
Ulcerative (classic)	~85%	Undermined violaceous borders; pretibial; severe pain; rapidly expanding
Bullous (atypical)	Rare	Superficial bullae on upper extremities/face; associated with hematologic malignancy
Pustular	Uncommon	Discrete painful pustules; strongly associated with inflammatory bowel disease (IBD); no frank ulceration
Vegetative	Rare	Warty/vegetative plaques; non-painful; no systemic association; responds to local therapy
Peristomal	Uncommon	Around stoma sites; almost always associated with IBD; treat underlying disease
Extracutaneous	Rare	Lungs (nodules/cavities), eyes (peripheral ulcerative keratitis, scleritis), joints, liver, spleen



(**A**) (**B**)

Figure 5: Pyoderma gangrenosum. (**A**) Early hemorrhagic ulcer on the lower leg [15]. Pyoderma gangrenosum image. DermNet®. December 5, 2023. Accessed September 10, 2026. https://dermnetnz.org/imagedetail/1396-pyoderma-gangrenosum. (**B**) Pyoderma gangrenosum on the finger [16]. Pyoderma gangrenosum image. DermNet®. December 5, 2023. Accessed September 10, 2026. https://dermnetnz.org/imagedetail/1384-pyoderma-gangrenosum.

Associated Conditions

- **Inflammatory bowel disease (20%–30%).** The most common association; occurs with both ulcerative colitis and Crohn's disease; peristomal PG occurs mostly in patients with IBD
- **Inflammatory arthritis (~10%).** Rheumatoid arthritis and seronegative spondyloarthropathies, including psoriatic arthritis and ankylosing spondylitis
- **Hematologic disease (~10%).** Monoclonal gammopathy of undetermined significance (MGUS), multiple myeloma, myelodysplastic syndrome, and leukemia, with AML particularly linked to the bullous variant
- **Other associations.** Solid tumors, hepatitis C, HIV, thyroid disease, and solid organ transplantation

Overall, 50% to 70% of PG patients have an underlying systemic disease, reinforcing the need for systemic evaluation at diagnosis.

Diagnosis

PG is a diagnosis of exclusion. The 2018 Delphi consensus criteria require 1 major criterion—biopsy showing a neutrophilic infiltrate—together with minor criteria that include pathergy, a history of IBD or inflammatory arthritis, characteristic ulcer morphology (undermined border, cribriform scarring), rapid spread, and rapid response to immunosuppression [13].

Key Differential Diagnoses to Exclude.

Category	Conditions to Exclude	Workup
Infectious	Ecthyma gangrenosum, necrotizing fasciitis, deep fungal infection (*Aspergillus*, *Mucor*), atypical mycobacteria	Wound cultures—bacterial, fungal, mycobacterial

Vascular	Calciphylaxis, antiphospholipid syndrome, arterial/venous ulcers, cholesterol emboli	CBC, CMP, ESR/CRP, SPEP, ANA, ANCA, C3, C4 antiphospholipid antibodies, blood cultures
Vasculitis	GPA (Wegener's), polyarteritis nodosa, leukocytoclastic vasculitis	Same laboratory panel as for Vascular
Malignancy	Squamous cell carcinoma, cutaneous lymphoma, Kaposi sarcoma	Skin biopsy
Other	Factitial ulcers, halogenoderma, brown recluse spider bite, lichen planus	Clinical correlation; colonoscopy or imaging if IBD or extracutaneous disease is suspected

ANA, antinuclear antibody; ANCA, antineutrophil cytoplasmic antibody; CBC, complete blood count; CMP, complete metabolic panel; ESR/CRP, erythrocyte sedimentation rate/C-reactive protein; GPA, granulomatosis with polyangiitis; SPEP, serum protein electrophoresis.

Treatment

Aggressive surgical debridement should be avoided because it can trigger further ulcer expansion (pathergy). Systemic immunosuppression is the mainstay of treatment, supported by appropriate wound care [14].

First-line systemic therapy

- Prednisone 0.5–1 mg/kg/day: rapid onset but a high side-effect burden
- Cyclosporine 3–5 mg/kg/day: an effective alternative requiring monitoring of blood pressure and renal function
- These agents can be combined for severe or refractory disease

Biologics for refractory disease

- Tumor necrosis factor alpha inhibitors: infliximab (best evidence), adalimumab
- Interleukin-1 inhibitors: anakinra, canakinumab (particularly for autoinflammatory PG)
- Janus kinase inhibitors: tofacitinib, upadacitinib (emerging evidence)
- Interleukin-12/23 and interleukin-23 inhibitors: ustekinumab, guselkumab

Topical and adjunctive measures

- Potent topical corticosteroids (clobetasol) or topical tacrolimus 0.1%
- Intralesional triamcinolone at the ulcer border
- Steroid-sparing agents: mycophenolate mofetil, azathioprine, methotrexate, dapsone
- Intravenous immunoglobulin for severe or refractory disease; hyperbaric oxygen as an adjunct

Clinical Course

PG typically follows a chronic, relapsing–remitting course. Approximately 50% of patients achieve remission, but recurrence is common, and healing characteristically leaves cribriform ("wrinkled paper") or atrophic scarring. The bullous variant carries higher mortality, reflecting its association with underlying hematologic disease. Long-term monitoring should address infection risk, bone density, renal and hepatic function, and the risk of immunosuppression-related malignancy.

5. Fixed Drug Eruption (Figure 6)

> ***Illustrative case:*** *A 42-year-old develops a rash 2 hours after a dose of trimethoprim–sulfamethoxazole. The key clue: The patient had a rash in the exact same location the previous year.*

Definition and Diagnostic Hallmark

Fixed drug eruption (FDE) is a distinctive cutaneous adverse drug reaction marked by recurrence of lesions at the same anatomical site with every re-exposure to the causative agent. Same-site recurrence with drug re-exposure is the single most reliable clinical clue to the diagnosis and is more diagnostic than morphology or histology alone [17].

Mechanism

FDE is a type IV, T-cell-mediated delayed hypersensitivity reaction, driven specifically by CD8+ tissue-resident memory T cells (TRM). Onset after re-exposure is rapid, occurring within 30 minutes to 8 hours [18].

Pathogenesis: Local memory T cells drive recurrence at the exact same site [18]

- CD8+ TRM cells persist in the epidermis at the site of a prior lesion long after clinical resolution.
- Re-exposure to the drug (or a cross-reactive agent) reactivates these TRM cells within hours, releasing interferon-gamma, perforin, and granzyme B.
- Cytotoxic signaling drives basal keratinocyte vacuolization and apoptosis, producing the dusky, sharply demarcated plaque.
- Because TRM cells are locally fixed within the tissue, the reaction reappears at the identical site with every future exposure.

Common Causative Agents

Class	Examples
Antibiotics	Trimethoprim-Sulfamethoxazole, tetracyclines, penicillins, quinolones
Nonsteroidal anti-inflammatory drugs (NSAIDs)	Ibuprofen, naproxen, piroxicam, mefenamic acid
Analgesics	Acetaminophen—increasingly reported
Others	Barbiturates, carbamazepine, phenolphthalein (in laxatives), oral contraceptives

Clinical Features

The morphology, distribution, and time course of FDE are well characterized [17].

- **Morphology:** A sharply demarcated, round-to-oval erythematous or violaceous macule or plaque that may blister; residual slate-gray to brown post-inflammatory pigmentation is characteristic.
- **Sites:** The lips, genitalia (especially the glans penis), acral areas, and trunk are most common; lesions may be solitary or multiple.

- **Symptoms:** Burning or itching predominates over pain, and systemic symptoms are typically absent, which helps distinguish FDE from Stevens–Johnson syndrome/toxic epidermal necrolysis (SJS/TEN).



(**A**) (**B**)

Figure 6: Fixed drug eruption. (**A**) Sharply demarcated, dusky violaceous plaque on the back. Drug reaction image. DermNet®. December 5, 2023. Accessed September 10, 2026. https://dermnetnz.org/imagedetail/8239-drug-reaction. (**B**) Bullous (blistering) fixed drug eruption [19]. Fixed drug eruption image. DermNet®. December 5, 2023. Accessed September 10, 2026. https://dermnetnz.org/imagedetail/11572-fixed-drug-eruption.

Diagnosis and Management

The diagnosis is largely clinical, anchored by the history of an identical lesion recurring at the same site with prior exposure to the same or a chemically related drug. Management centers on identifying and permanently discontinuing the causative agent; topical corticosteroids can be used for symptomatic relief of an acute lesion. Because lesional TRM cells persist for years, often after the pigmentation has faded, patients should be counseled to avoid the culprit drug and structurally related compounds lifelong.

Key Teaching Points

- Same-site recurrence, not lesion morphology, is the diagnostic hallmark.
- FDE is a type IV (T-cell-mediated) reaction, not a type I IgE-mediated reaction—it should not be mistaken for urticaria or anaphylaxis.
- NSAIDs are themselves a recognized cause of FDE, not a treatment for it.
- Unlike Sweet's syndrome or reactive processes associated with hematologic malignancy, an isolated FDE does not, by itself, warrant an oncologic workup.

Comparative Summary

Although these 5 conditions arise through distinct mechanisms, comparing them side-by-side highlights the diagnostic value of tempo, distribution, and systemic association.

Condition	Key Trigger/Association	Characteristic Distribution	Tempo	First-Line Treatment
Keratoderma blennorrhagica	Reactive arthritis (chlamydia, gastrointestinal pathogens); HLA-B27	Palms, soles; glans penis (circinate balanitis)	Days to weeks after infection; may persist weeks to months	Treat underlying infection; topical steroids/keratolytics
Erythema marginatum	Acute rheumatic fever (post-streptococcal)	Trunk, proximal limbs; spares face	Evanescent—hours	Treat rheumatic fever; anti-inflammatory therapy
Sweet's syndrome	Infection, malignancy (esp. AML), drugs (G-CSF, trimethoprim-sulfamethoxazole)	Face, neck, upper extremities	Abrupt onset	Systemic corticosteroids
Pyoderma gangrenosum	IBD, inflammatory arthritis, hematologic disease	Pretibial (classic); peristomal; varies by variant	Chronic, relapsing–remitting	Corticosteroids or cyclosporine; biologics if refractory
Fixed drug eruption	Drug re-exposure (antibiotics, NSAIDs, analgesics)	Lips, genitalia, acral skin, trunk—same site every time	30 minutes to 8 hours after re-exposure	Discontinue and avoid culprit drug

Diagnostic Pearls

- Tempo distinguishes look-alikes: Erythema marginatum resolves within hours, while erythema multiforme lesions persist for days. This changes the differential from rheumatic fever to a drug- or HSV-triggered reaction.
- Neutrophilic dermatoses form a spectrum: Sweet's syndrome and PG both feature a sterile neutrophilic infiltrate and can coexist or overlap with malignancy or IBD; PG is distinguished by ulceration and pathergy, Sweet's syndrome by tender plaques and fever.
- Site fidelity is diagnostic: Fixed drug eruption characteristically recurs at the identical anatomical location with each exposure, a pattern virtually pathognomonic for FDE.
- Palmoplantar pustular lesions have a differential beyond psoriasis: In a sexually active young adult with palmoplantar lesions and a painless genital lesion, reactive arthritis and keratoderma blennorrhagica should be considered alongside pustular psoriasis and syphilis.
- Cutaneous disease can be the presenting sign of malignancy: Both Sweet's syndrome and the bullous variant of PG have well-described associations with acute myeloid leukemia.

Conclusion

Across these 5 conditions, careful attention to a rash's morphology, distribution, tempo, and recurrence pattern allows the clinician to move efficiently from cutaneous finding to systemic diagnosis. Keratoderma blennorrhagica and erythema marginatum illustrate how the skin can encode a preceding infection and point toward reactive arthritis or acute rheumatic fever, respectively. Sweet's syndrome and pyoderma gangrenosum demonstrate a shared neutrophilic dermatosis pathophysiology that links the skin to inflammatory bowel disease, inflammatory arthritis, and hematologic malignancy. Fixed drug eruption demonstrates the unique diagnostic power of a focused medication and recurrence history. Together, these cases reinforce that dermatologic pattern recognition, paired with a directed systemic evaluation, remains one of the most efficient diagnostic tools available at the bedside.

Funding: This research received no external funding.

Acknowledgments: The author used Claude for content assistance, specifically to create an initial draft from prior personal presented material. The manuscript was reviewed and the author revised the material generated and takes full responsibility for the content of this publication.

Conflicts of Interest: The author declares no conflicts of interest.

References

1. Wu, I.B.; Schwartz, R.A. Reiter's syndrome: The classic triad and more. *J. Am. Acad. Dermatol.* **2008**, *59*, 113–121. [CrossRef] [PubMed]
2. Carter, J.D.; Hudson, A.P. Reactive arthritis: Clinical aspects and medical management. *Rheum. Dis. Clin. N. Am.* **2009**, *35*, 21–44. [CrossRef] [PubMed]
3. Rein, M.F. Keratoderma blennorrhagicum [Image]. Public Health Image Library (PHIL), ID #6950. Centers for Disease Control and Prevention. Available online: https://phil.cdc.gov/details.aspx?pid=6950 (accessed on 4 September 2026).
4. Maurer, T.A.; Berger, T.G. Keratoderma blennorrhagicum: Classic. U.S. Department of Veterans Affairs. Available online: https://www.hiv.va.gov/provider/image-library/dermatological.asp?post=1&slide=6 (accessed on 4 September 2026).
5. Gewitz, M.H.; Baltimore, R.S.; Tani, L.Y.; Sable, C.A.; Shulman, S.T.; Carapetis, J.; Remenyi, B.; Taubert, K.A.; Bolger, A.F.; Beerman, L.; et al. Revision of the Jones criteria for the diagnosis of acute rheumatic fever in the era of Doppler echocardiography: A scientific statement from the American Heart Association. *Circulation* **2015**, *131*, 1806–1818. [CrossRef] [PubMed]
6. Erythema marginatum. DermNet. Available online: https://dermnetnz.org/imagedetail/8033-erythema-marginatum (accessed on 3 September 2026).
7. Erythema marginatum. SkinCareGuide.ca. Available online: https://www.skincareguide.ca/glossary/e/erythema_marginatum.html (accessed on 4 September 2026).
8. King, C. Erythema multiforme. *DermNet*, February 2022. Available online: https://dermnetnz.org/topics/erythema-multiforme (accessed on 3 September 2026).
9. Erythema multiforme. SkinCareGuide.ca. Available online: https://www.skincareguide.ca/glossary/e/erythema_multiforme.html (accessed on 4 September 2026).
10. Cohen, P.R. Sweet's syndrome—A comprehensive review of an acute febrile neutrophilic dermatosis. *Orphanet J. Rare Dis.* **2007**, *2*, 34. [CrossRef] [PubMed]
11. von den Driesch, P. Sweet's syndrome (acute febrile neutrophilic dermatosis). *J. Am. Acad. Dermatol.* **1994**, *31*, 535–556. [CrossRef] [PubMed]
12. Nadarajah, N.; Imran, F. Acute febrile neutrophilic dermatosis (Sweet syndrome). *DermNet*, Updated February 2025. Available online: https://dermnetnz.org/topics/acute-febrile-neutrophilic-dermatosis (accessed on 3 September 2026).
13. Maverakis, E.; Ma, C.; Shinkai, K.; Fiorentino, D.; Callen, J.P.; Wollina, U.; Marzano, A.V.; Wallach, D.; Kim, K.; Schadt, C.; et al. Diagnostic criteria of ulcerative pyoderma gangrenosum: A Delphi consensus of international experts. *JAMA Dermatol.* **2018**, *154*, 461–466. [CrossRef] [PubMed]
14. Maronese, C.A.; Pimentel, M.A.; Li, M.M.; Genovese, G.; Ortega-Loayza, A.G.; Marzano, A.V. Pyoderma gangrenosum: An updated literature review on established and emerging pharmacological treatments. *Am. J. Clin. Dermatol.* **2022**, *23*, 615–634. [CrossRef] [PubMed]
15. Oakley, A.; Dhawan, G. Pyoderma gangrenosum. *DermNet*, March 2022. Available online: https://dermnetnz.org/topics/pyoderma-gangrenosum (accessed on 3 September 2026).
16. Pyoderma gangrenosum. DermNet. Available online: https://dermnetnz.org/imagedetail/1384-pyoderma-gangrenosum (accessed on 4 September 2026).
17. Tekely, E.; Kdziela-Wypyska, G.; Chodorowska, G. Fixed drug eruption—Pathogenesis and diversity of clinical manifestations. *Przegl. Dermatol.* **2012**, *99*, 252–258.
18. Shiohara, T. Fixed drug eruption: Pathogenesis and diagnostic tests. *Curr. Opin. Allergy Clin. Immunol.* **2009**, *9*, 316–321. [CrossRef] [PubMed]
19. Oakley, A.; Condon, C.; Nixon, R. Fixed drug eruption. *DermNet NZ*, March 2021. Available online: https://dermnetnz.org/topics/fixed-drug-eruption (accessed on 3 September 2026).