REVIEW

Inpatient Evaluation and Management of Generalized Pustular Dermatoses

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Abstract

Purpose of Review The purpose of this paper is to discuss the evaluation and management of the most encountered pustular dermatoses in the inpatient setting.

Recent Findings Generalized pustular psoriasis (GPP), acute generalized exanthematous pustulosis (AGEP), and acneiform eruptions are three pustular dermatoses encountered in the inpatient setting. New insights into the pathophysiology of these diseases are guiding the investigation of novel treatments for pustular dermatoses.

Summary Although cases of pustular dermatoses are often benign and self-limited, careful evaluation and management are indicated as severe cases may pose legitimate health risks and require systemic therapy. Biologic agents are playing a larger role in the management of pustular dermatoses such as GPP.

Keywords Pustular dermatoses · Inpatient · Generalized pustular psoriasis · Acute generalized exanthematous pustulosis

Introduction

A pustule is a fluid-filled raised lesion on the skin enclosing an accumulation of neutrophils. To be considered a pustule, the lesion must be colored white/yellow from inception. This differentiates the pustule from a vesicle which may become turbid over time as it is filled with leukocytes [1]. This difference is key during initial evaluation as there are separate differential diagnoses of these two primary lesions. This review article discusses the evaluation and management of pustular eruptions in the inpatient setting, with a particular focus on two of the most commonly encountered pustular dermatoses: generalized pustular psoriasis and acute generalized exanthematous pustulosis. In addition, acneiform drug eruptions are also discussed as this condition presents frequently in the inpatient setting with pustular lesions.

Ruling Out Infection

It is important to first rule out primary infection as a potential underlying cause for a pustular eruption. Some examples to consider that may present with pustules on the skin include infectious folliculitis, bullous impetigo, or cutaneous fungal infections such as from dermatophytes or candida. Rarely, conditions such as septic/infectious vasculitis (e.g., meningococcemia and gonococcemia) and herpes infections (e.g., herpes simplex or herpes zoster) may present with pustules and need to be considered.

In 2014, the Infectious Diseases Society of America (IDSA) published practice guidelines for the evaluation and management of skin and soft tissue infections (SSI) [2]. Per the IDSA guidelines, culture and sensitivity of exudate or pus ought to be performed in moderate-severe cases of SSI [2]. These tests are helpful to investigate for various microorganisms (e.g., *Staphylococcus aureus* and group A streptococcus). Other studies can also be considered to rule out infections including skin biopsy, tissue culture, and viral testing.

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Generalized Pustular Psoriasis

Generalized pustular psoriasis (GPP) is a rare, heterogenous disease with characteristic abrupt flares of erythema and sterile, neutrophilic pustules generalized on the skin [3]. Prevalence ranges from 0.02 to 1.4 per 10,000 people with the highest rates reported in Germany and South Korea [3–7]. Onset of GPP is typically in the fifth or sixth decade of life [6, 8, 9]. GPP is rare in children, but when it occurs in this population, it may present more severely [3]. Most commonly, patients may have periods of clear skin between flares, but persistent disease can occur [3, 10••, 11].

The clinical features of GPP include variable-sized, macroscopically visible pustules, typically on erythematous skin [3]. Crusts and scales may also be present [3]. GPP typically does not affect acral surfaces and can occur with or without concurrent or previous history of plaque psoriasis [3]. The pustules may coalesce into larger lesions often referred to as "lakes of pus" which may last for multiple days or weeks [3]. Systemic symptoms such as fever, chills, arthralgia, and malaise may occur and these patients are often hospitalized as a consequence. If flares are unmanaged, patients may be at risk of complications such as multisystem organ failure, infections, systemic capillary leak syndrome, liver disease, acute renal failure, heart failure, arthritis, sepsis, and death [3, 12].

The etiology of GPP is incompletely understood [3]. The general consensus is that the disease is distinct from plaque psoriasis [3]. There is considerable evidence pointing to the involvement of IL-36 signaling in the pathogenesis of GPP. Investigational studies have demonstrated loss-of-function mutations of the IL36 antagonist gene (IL36RN) and overexpression of IL-36 cytokines in skin lesions [13–17]. Additionally, mutations in the following genes are thought to be potentially associated with GPP: IL36N, CARD14, AP1S3, and SERPINA3 [3].

Triggers for GPP flares may include infection, pregnancy, hypocalcemia, and medication withdrawal. However, GPP may also erupt idiopathically [3]. The following medications have been associated with flares of GPP: systemic corticosteroids, biologics (TNFα inhibitors, ustekinumab, and secukinumab), lithium, amoxicillin, ceftriaxone, oxacillin, terbinafine, rituximab, codeine, pegylated interferon-alpha-2b, and cyclosporine [3, 18–25].

Making the Diagnosis

The diagnosis of GPP is a clinical diagnosis and may be made after taking a concordant medical history and observing characteristic findings on physical examination.

However, laboratory evaluations and skin biopsy with histopathologic evaluation are helpful for establishing the diagnosis and may be useful in guiding therapy. Both the European Rare and Severe Psoriasis Expert Network (ERASPEN) and the Japanese Dermatologic Association have created diagnostic criteria that incorporate both clinical and histopathologic features; however, no current consensus exists in the USA. Per the Japanese Dermatologic Society, a diagnosis of GPP can be made if a patient exhibits four findings: (1) systemic symptoms, (2) systemic or extensive flush with multiple sterile pustules, (3) neutrophilic subcorneal pustules histopathologically, and (4) recurrence of the typical clinical and histologic findings. GPP could be suspected if the patient exhibits 2 or 3 of these findings [26].

Similarly, ERASPEN guidelines state that a diagnosis of GPP requires the presentation of primary, sterile pustules on non-acral skin that can be appreciated macroscopically [10••]. ERASPEN also provides subclassification descriptors that include three factors: (1) the presence of systemic inflammation, (2) the presence of plaque psoriasis, and (3) chronicity/temporality (i.e., relapsing vs persistent) [10••].

The differential diagnosis for GPP includes AGEP, subcorneal pustular dermatosis, IgA pemphigus, erythrodermic or unstable plaque psoriasis, and infectious disease (e.g., bullous/non-bullous impetigo, abscesses, fungal infections).

Testing Overview

During flares, levels of systemic markers of inflammation such as erythrocyte sedimentation rate and C-reactive protein may be elevated [3]. Additional lab findings include anemia, thrombocytosis, hypoalbuminemia, electrolyte derangement, hypocalcemia, and leukocytosis with/without eosinophilia [3]. The histopathologic features of GPP include the migration of neutrophils into the epidermis with the formation of aggregates between degenerated keratinocytes (Kogoj's spongiform pustules). Certain psoriasiform histologic changes may also be appreciated: parakeratosis, elongation of rete ridges, and a superficial perivascular mononuclear cell infiltrate [3].

Upon initial presentation, it may be difficult to distinguish between GPP and acute generalized exanthematous pustulosis. A recent study found that staining for plasmacytoid dendritic cells (IFN-α/β producer) and MxA (IFN-α/β-inducible protein) could potentially assist in making the distinction as these findings significantly favor a diagnosis of pustular psoriasis [27].

Management

Unfortunately, due to the paucity of clinical trials, there is no standardized management of GPP as of 2023 [3]. Large clinical trials are not considered feasible due to the rarity of the disease [3]. However, in countries such as Japan, Thailand, and Taiwan, several biologic agents (TNFα, IL17, and IL-23 inhibitors) have been approved for the treatment of GPP based on data from smaller clinical trials (Table 1) [12, 28••, 29–32]. These agents are not approved for the management of GPP in the USA and Europe.

Management of GPP in the USA and Europe has largely consisted of agents such as methotrexate, cyclosporine, retinoids, and biologic agents [26, 30, 33•, 34]. The US Medical Board of the National Psoriasis Foundation published recommendations in 2012 and specifically recommended cyclosporine, acitretin, methotrexate, or infliximab as first-line therapy [33•]. Although dosing recommendations for these agents have not been established, it can be helpful to note how these agents have been used previously in GPP cases. In a Chinese retrospective analysis of nine patients with GPP between 2016 and 2018, combination therapy with acitretin (0.5 mg/kg/day) and glycyrrhizin (Chinese herbal medicine) was administered for 2 weeks in what was considered the "acute" phase of management [35]. The acute phase was followed by tapering of acitretin (20–30 mg/day) [35]. Doses as high as 1.0 mg/kg/day for a duration of 4–8 weeks have also been reported [36]. An alternative retinoid, etretinate, has also been used at a starting dose of starting dose of 1 mg/kg/day [37]. Cyclosporine has been successfully utilized at doses ranging from 1 to 2 mg/kg/day for 2 to 12 months [38].

Second-line treatments for GPP have generally included etanercept, adalimumab, psoralen plus ultraviolet-A (PUVA)

phototherapy, and topical therapy (e.g., corticosteroids, tacrolimus) [33•]. Fortunately, targeted treatments for the treatment of GPP are on the horizon. Spesolimab, a humanized anti-interleukin-36 (IL-36) receptor monoclonal antibody, may be a potentially valuable tool in the long-term management of GPP. Currently, spesolimab is the only drug with FDA approval for the treatment of GPP in the USA. The drug is administered intravenously over 90 min with a 900-mg initial dose and the option for a supplemental 900-mg dose 1 week later. The IL-36 pathway is believed to be the key proinflammatory pathway involved in GPP [28••, 39]. In the Effisayil 1 study, use of spesolimab resulted in a higher incidence of pustule clearance than placebo [28••]. After 1 week, 43% patients in the treatment group (received single 900-mg dose) had a Generalized Pustular Psoriasis Physician Global Assessment (GPPGA) total score of 0 or 1 compared with 11% in the placebo group. Regarding pustulation specifically, 54% of the patients in the treatment group demonstrated pustule clearance compared with 6% in the placebo group [28••]. However, spesolimab also resulted in an increased incidence of infection and systemic drug reactions. In the treatment group, 17% of the patients experienced an infection by week one compared to 6% of the placebo group [28••]. Two patients had a drug reaction with eosinophilia and systemic symptoms (DRESS) and one of these patients experienced drug-induced hepatic injury [28••]. A follow-up trial, the Effisayil 2 study, is investigating maintenance therapy with intermittent injections of spesolimab in patients with a documented history of GPP [39]. The primary endpoint in this study is time to first GPP flare [39].

Additional biologics targeting the IL-1-/IL-36 inflammatory pathway have been used in the management of GPP. Specifically, anakinra (IL-1 receptor antagonist),

Table 1 Biologic therapies approved for GPP. *TNFαi* tumor necrosis alpha inhibitor, *IL-12/23i* interleukin-12/23 inhibitor, *IL-17i* interleukin-17 inhibitor, *IL-17RAi* interleukin-17 receptor A inhibitor, *IL-23i* interleukin-23 inhibitor, *IL-36Ri* interleukin-36 receptor inhibitor

Biologic name	Dosing	Countries approved	Mechanism of action
Infliximab	5 mg/kg administered as a slow infusion over 2 h. Additional doses administered at weeks 2, 6, and every 8 weeks thereafter, as needed	Japan	TNFαi
Adalimumab	S.c. injection of 80 mg at week 0 followed by 40 mg every 2 weeks thereafter	Japan	TNFαi
Ustekinumab	S.c. injections of 45 mg at weeks 0 and 4 and every 12 weeks thereafter	Japan	IL-12/23i
Secukinumab	S.c. injections of 300 mg on weeks 0, 1, 2, 3, and 4 and every 4 weeks thereafter (may be decreased to 150 mg)	Japan	IL-17i
Ixekizumab	S.c. injections of 160 mg at week 0, followed by 80 mg at weeks 2, 4, 6, 8, 10 and 12, then 80 mg every 4 weeks thereafter	Japan	IL-17i
Brodalumab	S.c. injections of 210 mg at weeks 0, 1, 2 and then every 2 weeks thereafter	Japan, Taiwan, and Thailand	IL-17RAi
Guselkumab	S.c. injections of 100 mg at week 0, week 4, and every 8 weeks thereafter	Japan	IL-23i
Risankizumab	S.c. injections of 150 mg at week 0 and week 4 and every 12 weeks thereafter	Japan	IL-23i
Spesolimab	S.c. injection of 900 mg at week 0 and an optional 900-mg injection 1 week later	USA and European Union	IL-36Ri

canakinumab, and gevokizumab (both anti-IL-1β monoclonal antibodies) demonstrated favorable results in case reports and case series [40–43].

One Japanese retrospective cohort analysis of 1516 patients with GPP found better mortality outcomes with biologic therapy when compared to oral agents (methotrexate etretinate, cyclosporine) or systemic corticosteroids [44]. In addition, it was noted that, among those treated with a biologic agent, IL-17 inhibitors demonstrated equal in-hospital mortality and morbidity to TNF inhibitors [44]. In general, systemic corticosteroids have been used less frequently due to the concern for an increased risk of rebound flare with tapering. However, in a recent retrospective cohort study of 1970 psoriasis patients receiving corticosteroids, only 1.42% experienced a flare [45].

In the pregnant population, data and general recommendations are significantly limited. However, a few specific medications have been utilized and reported. Interestingly, cyclosporine 2.5–5 mg/kg per day has been used successfully despite being generally contraindicated in pregnant women [46]. Benefits of cyclosporine in this setting of GPP are considered to outweigh the potential risks of the medication [46]. Additional medications utilized include corticosteroids, TNFαi, and granulocyte and monocyte adsorption apheresis (GMA) [46]. GMA is considered a relatively safe option; however, data regarding use of GMA in pregnancy is severely limited [46].

Acute Generalized Exanthematous Pustulosis

Acute generalized exanthematous pustulosis (AGEP) is a cutaneous eruption, usually due to a medication, of small, sterile, non-follicular pustules overlying an erythematous base. AGEP is an uncommon condition with a reported incidence of 1–5 patients per million per year [47]. Cutaneous symptoms typically start in the main folds of the body (axillae, submammary folds, and inguinal folds) and spread to the trunk and limbs within hours to a few days [48]. Roughly one-fifth of patients with AGEP will experience mucosal involvement [48]. Although the clinical course is usually benign, evidence of systemic involvement is present in approximately 20% of cases [48]. AGEP with systemic involvement has mortality rates as high as 5% [48, 49]. After the underlying cause is addressed, symptoms usually resolve within a couple of weeks [48, 49]. As the pustules resolve, they leave behind collarettes of superficial desquamation. This eruption may also be localized to a single area of the body. When this occurs, the term "acute localized exanthematous pustulosis" (ALEP) is more appropriate [48, 50].

Over 90% of cases of AGEP are associated with medications such as aminopenicillins, sulphonamides, quinolones, pristinamycin, hydroxychloroquine, diltiazem, and terbinafine [48–52, 53•]. It is estimated that the time from causative drug ingestion to reaction onset is less than 2 days [48, 53•] Other associated causes include infections such as E.coli, chlamydia, coxsackie, parvovirus, EBV, cytomegalovirus, mycoplasma, echinococcosis and coccidiomycosis [52, 54–58]. Additionally, spider bites, psoralen combined with ultraviolet A light therapy, herbal medications, mercury, lacquer, xenobiotics, and venoms have reportedly caused AGEP [59–63]. Iodinated contrast is also a reported trigger and of particular relevance when evaluating patients with AGEP in the inpatient setting [64].

AGEP is described as a sterile, T-cell mediated, neutrophilic inflammatory response involving drug-specific CD4 + T cells, cytotoxic CD8 + T cells, inflammatory cytokines, and chemokines [65–67]. T helper 17 cells and genetic variations in the interleukin-36 receptor antagonist gene (IL-36RN) have been implicated in the pathogenesis of AGEP [68, 69].

Diagnosis

The differential diagnosis of AGEP includes generalized pustular psoriasis, Sweet syndrome, subcorneal pustulosis (Sneddon-Wilkinson syndrome), drug rash with eosinophilia and systemic symptoms (DRESS), pustular vasculi-tis, immunobullous disease, cutaneous fungal infections, and bullous impetigo [70, 71]. It can be difficult to differentiate between AGEP and GPP when they initially present. A diagnosis of GPP is favored when a patient presents with a history of psoriasis, lack of drug exposure, a longer duration of symptoms (i.e., pustules, fever), or histologic findings of acanthosis and papillomatosis [72].

The EuroSCAR study produced a scoring system with diagnostic criteria to assist clinicians in making the diagnosis of AGEP [53•, 71]. The criteria include morphology of the cutaneous eruption, clinical course, and histopathologic findings (see Table 2).

As seen in Table 2, the principal laboratory finding consistent with AGEP is a neutrophilic-predominant leukocytosis on CBC (PMN > 7000/μL). Histologic findings include spongiform subcorneal pustules with/without intraepidermal pustules [73]. Necrosis of solitary keratinocytes, clear edema of the papillary dermis, and a superficial-to-mid-dermal infiltrate of neutrophils may be appreciated [73]. Additionally, eosinophils may be present in pustules or in the dermal layer [73].

In a retrospective review of 340 cases of AGEP, 7–9% of patients demonstrated evidence of hepatic involvement and/ or kidney insufficiency [74]. Hypocalcemia was also found in 65.7% of the patients [74].

Management

Many consider the priority in AGEP management to be the removal of the causative agent and medical treatment to be

Table 2 Diagnostic score for acute generalized exanthematous pustulosis from EuroSCAR study score interpretation: 0 no; 1–4 possible; 5–7 probable; 8–12 definitive

Variable			Score
Morphology			
Pustules		Typical morphology	+2
		Compatible with disease	+1
		Insufficient	0
Erythema		Typical morphology	+2
		Compatible with disease	+1
		Insufficient	0
Distribution/pattern		Typical morphology	+2
		Compatible with disease	+1
		Insufficient	0
Course			
Mucosal involvement		Yes/no	−2/0
Acute onset		Yes/no	0/−2
Resolution (<15 days)		Yes/no	0/−2
Fever > 38.5 °C		Yes/no	+1/0
Polymorphonuclear cells > $7000/mm^2$		Yes/no	+1/0
Histology			
Other disease		Present	−10
Not representative		Present	0
Exocytosis of PMN cells		Present	+1
Subcorneal and/or intraepidermal non-spongiform or NOS pustule(s) with papillary edema or subcorneal and/or intraepidermal spongiform or NOS pustules without papillary oedema		Present	+2
Spongiform subcorneal and/or intraepidermal pustules with papillary oedema		Present	+3

unnecessary [75, 76]. However, agents such as topical and oral corticosteroids (e.g., prednisone) have been used successfully in the management of AGEP. High-potency topical steroids (class I and class II) are typically applied for 5 to 10 days during the acute phase to help alleviate symptoms [70, 76]. Oral steroids have generally been reserved for severe disease with signs of systemic involvement. However, this is not supported by clinical trials and has not been shown to shorten disease, and cases of steroid-induced AGEP have been reported in the literature [48, 77–79]. Lastly, antihistamines have also been used for symptom management, typically in combination with oral or topical steroids [79].

Acneiform Drug Eruptions

Although acneiform drug eruptions may be caused by various drugs (Table 3), our review focuses on targeted chemotherapy induced acneiform eruptions due to their prevalence in inpatient settings. In fact, the prototypical cutaneous adverse reaction to an epidermal growth factor (EGFR) inhibitor or an MEK inhibitor is the acneiform eruption [80–82]. These acneiform eruptions may cause pain, pruritus, and impairment of quality of life and may affect sensitive areas [83].

EGFR acneiform eruptions characteristically present as erythematous papules and pustules in areas with a high density of sebaceous glands. Unlike typical acne vulgaris, comedones are characteristically absent [84]. The upper trunk is frequently involved [85]. These eruptions typically peak 2–3 weeks after the treatment onset and subsequently transition to a less severe chronic phase [86–88].

The pathophysiology of an acneiform drug eruption is incompletely understood. However, it is known that direct

Table 3 Drugs associated with acneiform eruptions

Drug name or class
MEK inhibitors
EGFR inhibitors
Halogens (bromides, iodides)
Antibiotics (isoniazid, rifampicin, and tetracyclines)
Lithium
Anticonvulsants (phenobarbital, phenytoin)
Hormones (corticotropin, androgens, oral contraceptives)
Topical and systemic corticosteroids
Immunosuppressants
Vitamins (B_2, B_6, B_{12})

inhibition of EGFR by tyrosine kinase inhibitors or monoclonal antibodies downregulates levels of phosphorylated EGFR in basal and suprabasal keratinocytes and outer layer of hair follicles [89–93]. This results in diminished proliferation of basal keratinocytes, growth arrest and apoptosis of keratinocytes, premature and accelerated differentiation, and decreased migration of differentiating keratinocytes due to enhanced cell attachment. EGFR inhibitors also cause a cytokine-mediated (e.g., IL1, TNF-alpha) inflammatory response [85, 89, 94, 95].

Histopathologic findings of a biopsy of an acneiform eruption typically include a superficial perifolliculitis, neutrophilic infiltrate adjacent to hyperkeratotic and/or ectatic follicular infundibula, and a neutrophilic, florid, suppurative folliculitis with separation of the epithelial lining [96, 97].

Acneiform drug eruptions are not often considered life-threatening. However, undermanagement of lesions may lead to secondary infection and cutaneous sequelae such as postinflammatory hyperpigmentation, scarring, telangiectasias, and erythema [98, 99]. Superinfection should be considered if the following signs or symptoms are present: yellow discharge or crusting, lack of response to treatment, worsening of condition, pain upon palpation, or peripheral erythema.

Roughly 60% of patients with acneiform eruption from EGFR inhibitor use reported diminished quality of life with symptoms such as pain, burning, irritation, and pruritus. A retrospective study found that acneiform eruption severity positively correlated with median scores on the Skindex-16, a validated questionnaire that measures symptoms and perceptions of toxicity [99, 100].

Diagnosis

Diagnosis of an acneiform drug eruption is clinical and straightforward if the patient is demonstrating the typical morphology and distribution of the rash in the setting of drug treatment. Skin biopsies are typically unnecessary unless the patient does not respond to therapy. If superinfection is suspected, skin cultures of exudate should be obtained.

The National Cancer Institute's Common Terminology Criteria for Adverse Events (CTCAE) is the standard scale to grade the severity of cutaneous adverse reactions during cancer treatment [101]. Grade is determined by the extent of the lesions and by the presence or absence of systemic symptoms and infection. The grade of severity of acneiform rash can be subsequently used to guide management (Table 4).

Management

Preemptive Therapy (for Patients Starting EGFR Inhibitors)

Prophylactic oral antibiotics and topical corticosteroids for those patients starting EGFR inhibitors may be considered

Table 4 National Cancer Institute (NCI) Common Terminology Criteria for Adverse Event (CTCAE)s: acneiform rash v5.0, 2017 [102]

| Acneiform rash | Papules and/or pustules covering <10% of the body surface area, which may or may not be associated with symptoms of pruritus or tenderness | Papules and/or pustules covering 10 to 30% of the body surface area, which may or may not be associated with symptoms of pruritus or tenderness; associated with psychosocial impact; limiting instrumental ADL[a], papules and/or pustules covering >30% of the body surface area, with or without mild symptoms | Papules and/or pustules covering >30% of the body surface area with moderate or severe symptoms; limiting self-care ADL[a], associated with local superinfection with oral antibiotics indicated | Life-threatening consequences; papules and/or pustules covering any percent of the body surface area, which may or may not be associated with symptoms of pruritus or tenderness and are associated with extensive superinfection with intravenous antibiotics indicated | Death |

[a]*ADL* activities of daily living

[94]. On the same day as the initiation of EGFR therapy, prescribe a 6-week course of doxycycline 100 mg BID or minocycline 100 qd [94]. Alternatives include cephalosporins or trimethoprim-sulfamethoxazole (160 mg/800 mg twice daily) [94]. In addition, it is recommended to begin a low-potency topical steroid (e.g., hydrocortisone 2.5%) applied to the face and chest twice daily [94].

Additional measures to prevent an EGFR-induced acneiform eruption include avoidance of hot water (e.g., baths, hand washing, showers), antibacterial or perfumed soaps and detergents, skin irritants, and UV exposure [94]. Application of thick, moisturizing cream twice daily and broad-spectrum sunscreen is recommended [94].

Therapeutic Management

For low-grade acneiform rash (grades 1, 2), the causative chemotherapeutic drug may be continued, but topical corticosteroids in combination with oral tetracycline antibiotics (e.g., doxycycline 100 mg BID) for 6 weeks or more are recommended [94]. For a grade 3 rash or above, oral antibiotics are combined with a steroid taper starting with 0.5–1-mg/kg body weight for 7 days which is recommend [94]. Also, for a grade 3 rash, the causative chemotherapeutic drug maybe paused and skin cultures may be obtained [94]. Moderate- to high-potency topical steroids are often warranted for acneiform rashes. Agents such as dapsone and oral retinoids have also been used [82, 94]. For patients failing standard therapies, skin cultures should be considered as antibiotic-resistant bacteria can occur during treatment [103].

Table 5 Additional pustular dermatoses

Generalized
    IgA pemphigus
    Sneddon-Wilkinson
    DRESS
Localized pustular dermatoses
    Pustulosis palmaris et plantaris
    Acrodermatitis continua of Hallopeau
    Acne vulgaris
    Rosacea
    Perioral dermatitis
    Folliculitis
    Eosinophilic folliculitis (HIV, non-HIV-associated)
    Erosive pustular dermatosis
Infantile and neonatal pustular eruptions
    Erythema toxicum neonatorum
    Transient Neonatal Pustular Melanosis
    Neonatal and infantile acne
    Acropustulosis of infancy
    Eosinophilic pustular dermatosis of infancy

Other Sterile Eruptions

Several additional conditions are included in an extensive differential diagnosis for pustular dermatoses (Table 5).

Conclusions

Although several pustular dermatoses are benign and self-limited, it is important to be aware of the full differential as certain conditions may pose a true health risk to patient and require systemic therapy. It is important to understand that not all pustular dermatoses are infectious in etiology and that conditions such as GPP, AGEP, and acneiform eruptions are managed with significantly different agents and strategies.

Data Availability Data sharing is not applicable as no new data were created or analyzed in this study.

Compliance with Ethical Standards

Conflict of Interest Dr. Tina Bhutani is a principal investigator for trials sponsored by Abbvie, Castle, CorEvitas, Dermavant, Galderma, Mindera, and Pfizer. She has served as an advisor for Abbvie, Arcutis, Boehringer-Ingelheim, Bristol Myers Squibb, Janssen, Leo, Lilly, Novartis, Pfizer, Sun, and UCB. The remaining authors have nothing to disclose.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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•• Of major importance

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