Journal of Biosciences and Medicines, 2026, 14(3), 478-487
https://www.scirp.org/journal/jbm
ISSN Online: 2327-509X
ISSN Print: 2327-5081

# Adalimumab-Associated Acute Generalized Exanthematous Pustulosis with Psoriasiform Features: A Diagnostic Challenge

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**How to cite this paper:** Chimbi-Bru, M.P., Osorio-Contreras, K.S., Vanegas-Torres, L., Orozco-Pérez, V., Figueroa-Trujillo, K., Salazar-Arrieta, A., Lopez-Rincón, L., Vargas-Torres, M., Salazar-Castillo, J., Molinares-Diaz, L. and Castillo, N.Y.-D. (2026) Adalimumab-Associated Acute Generalized Exanthematous Pustulosis with Psoriasiform Features: A Diagnostic Challenge. *Journal of Biosciences and Medicines*, **14**, 478-487.
https://doi.org/10.4236/jbm.2026.143035

**Received:** February 8, 2026
**Accepted:** March 17, 2026
**Published:** March 20, 2026



## Abstract

**Background:** Acute generalized exanthematous pustulosis (AGEP) is a rare, severe cutaneous adverse reaction, most commonly induced by drugs. It is characterized by the abrupt onset of numerous sterile, non-follicular pustules on an erythematous background and is typically self-limited after withdrawal of the offending agent. Although antibiotics are the most frequent triggers, biologic therapies, including anti-tumor necrosis factor (TNF) agents, have increasingly been implicated. Distinguishing AGEP from pustular psoriasis or paradoxical psoriasis induced by anti-TNF therapy may be challenging due to overlapping clinical and histopathological features. **Case:** We report the case of a 51-year-old woman with rheumatoid arthritis treated with adalimumab and leflunomide, who presented with a 15-day history of generalized pustular eruption accompanied by malaise but no fever. Dermatological examination revealed widespread erythematous papules and pustules involving the trunk, back, extremities, and prominently the palms and soles, with marked involvement of the axillary folds. A punch biopsy from a dorsal lesion demonstrated histopathological findings consistent with acute generalized exanthematous pustulosis. Laboratory evaluation was unremarkable. Adalimumab was discontinued, and systemic cyclosporine was initiated, leading to complete resolution of the pustular eruption. During follow-up, the patient developed new psoriasiform lesions affecting the scalp and plantar surfaces, raising the possi-

bility of a clinical overlap between AGEP and paradoxical psoriasis secondary to anti-TNF therapy. **Conclusion:** This case highlights AGEP as a potential severe cutaneous adverse reaction to adalimumab and underscores the diagnostic complexity when psoriasiform features emerge during follow-up. Careful clinicopathological correlation and longitudinal assessment are essential to differentiate AGEP from paradoxical psoriasis, particularly in patients receiving biologic agents. Early recognition and prompt withdrawal of the causative drug remain the cornerstone of management.

## Keywords

Acute Generalized Exanthematous Pustulosis, Adalimumab, Anti-TNF Therapy, Drug-Induced Skin Eruption, Paradoxical Psoriasis, Pustular Dermatoses

## 1. Introduction

Acute generalized exanthematous pustulosis (AGEP) is an uncommon but potentially life-threatening severe cutaneous adverse reaction (SCAR), most often triggered by medications. It is characterized by the sudden eruption of numerous sterile, non-follicular pustules arising on a background of diffuse erythema, frequently associated with fever, leukocytosis, and neutrophilia. Histopathologically, AGEP is defined by subcorneal or intraepidermal pustules, marked papillary dermal edema, and a perivascular infiltrate rich in neutrophils and eosinophils [1] [2]. In most cases, AGEP follows a rapid and favorable course after prompt withdrawal of the offending drug; however, severe systemic involvement and fatal outcomes have been reported, underscoring the importance of early recognition [1].

Although antibiotics—particularly beta-lactams and macrolides—remain the most common causative agents, an increasing number of non-antibiotic drugs have been implicated in AGEP over the last two decades, including calcium channel blockers, antimalarials, and, more recently, biologic therapies [2] [3]. The growing use of targeted immunomodulatory agents has expanded the spectrum of drug-induced pustular eruptions, introducing new diagnostic challenges for clinicians and dermatopathologists alike.

Tumor necrosis factor-alpha (TNF-$\alpha$) inhibitors, such as adalimumab, infliximab, and etanercept, have revolutionized the treatment of chronic inflammatory diseases, including rheumatoid arthritis, inflammatory bowel disease, and psoriasis. Paradoxically, despite their anti-inflammatory properties, these agents have been associated with a wide range of immune-mediated cutaneous adverse events. Among these, paradoxical psoriasis represents the most well-recognized phenomenon, but pustular eruptions, eczematous dermatitis, and rare cases of AGEP have also been reported [4] [5]. The underlying immunopathogenesis remains incompletely understood, but current evidence suggests that TNF-$\alpha$ blockade may lead to dysregulated innate immune responses, with upregulation of interferon-$\alpha$ and

enhanced neutrophilic inflammation [6].

The distinction between AGEP and pustular or paradoxical psoriasis induced by anti-TNF therapy is particularly challenging. Clinically, both conditions may present with widespread pustules, prominent involvement of flexural areas and palmoplantar surfaces, and relative sparing of mucous membranes. Histopathological overlap further complicates the diagnostic process, as psoriasiform epidermal hyperplasia may be observed in AGEP, while neutrophilic pustules can also be a feature of pustular psoriasis [7]. Moreover, cases have been described in which patients initially diagnosed with AGEP subsequently develop chronic psoriasiform lesions, raising the question of whether these entities represent distinct conditions or points along a shared inflammatory spectrum triggered by biologic agents [8].

Accurate differentiation between AGEP and paradoxical psoriasis is of paramount clinical importance, as therapeutic strategies and prognostic implications differ substantially. AGEP is typically self-limited once the causative drug is discontinued, whereas paradoxical psoriasis may persist or even progress despite drug withdrawal and often requires long-term immunosuppressive or biologic therapy [5] [9]. Misclassification may therefore lead to inappropriate management and unnecessary exposure to systemic treatments.

In this context, we report a case of adalimumab-associated AGEP in a patient with rheumatoid arthritis, complicated by the subsequent development of psoriasiform lesions during follow-up. This case illustrates the diagnostic complexity at the interface between severe drug-induced pustular reactions and paradoxical immune phenomena related to anti-TNF therapy, emphasizing the need for careful clinicopathological correlation and longitudinal assessment in patients receiving biologic agents.

## 2. Case Report

A 51-year-old woman with a long-standing diagnosis of rheumatoid arthritis was referred to the dermatology service because of a generalized pustular eruption that had progressively evolved over a 15-day period. Her rheumatologic disease had been well controlled with adalimumab (40 mg subcutaneously every two weeks), initiated approximately 18 months before presentation, in combination with leflunomide 20 mg daily, which had been maintained at a stable dose for more than one year. The last adalimumab injection had been administered three weeks prior to symptom onset. No recent dose adjustments, treatment interruptions, vaccinations, infections, or introduction of new medications were reported.

The patient had no personal or family history of psoriasis, pustular dermatoses, or chronic inflammatory skin diseases.

The cutaneous eruption began abruptly and rapidly disseminated, accompanied by generalized malaise but notably without fever, chills, or systemic compromise. On physical examination, the patient was hemodynamically stable and afebrile. Dermatological evaluation revealed a widespread eruption composed of erythematous papules and numerous small, non-follicular pustules distributed over the trunk, back, and extremities. Lesions were particularly confluent within the axillary folds,

where clusters of pustules overlaid erythematous and mildly scaly plaques, producing a prominent intertriginous pattern (Figure 1). Marked palmoplantar involvement was also observed, with erythematous papules and sterile pustules affecting both plantar surfaces (Figure 2). Mucosal surfaces were spared, and no facial edema or lymphadenopathy was identified.

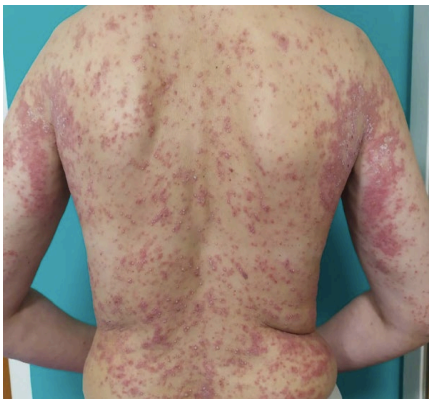

Figure 1. Clusters of pustular lesions overlying erythematous and scaly plaques involving the trunkand axillary folds.

Given the acute onset, morphology, and exposure to biologic therapy, a severe drug-induced pustular eruption was suspected.

Laboratory evaluation demonstrated leukocyte count of 7900 cells/mm$^3$ with absolute neutrophil count of 5400 cells/mm$^3$ and eosinophils of 180 cells/mm$^3$. C-reactive protein was mildly elevated at 8 mg/L. Renal and hepatic function tests were within normal limits (creatinine 0.8 mg/dL; AST 24 U/L; ALT 21 U/L). No leukocytosis, neutrophilia, or systemic inflammatory response was documented. Laboratory parameters remained stable when repeated during the peak phase of the eruption.

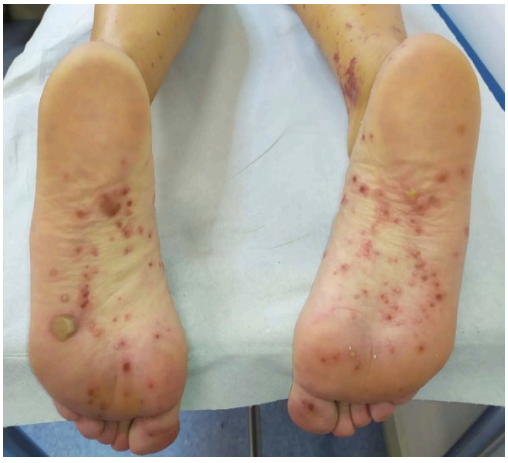

Figure 2. Pustules and erythematous papules involving the plantar surfaces.

To exclude infectious pustular dermatoses, bacterial cultures obtained from intact pustules were negative, and fungal examination showed no pathogenic organ-

isms. Blood cultures were not indicated due to absence of systemic symptoms. These findings supported a sterile inflammatory process.

A punch biopsy was obtained from an active lesion on the dorsal trunk to establish diagnostic confirmation and exclude pustular psoriasis. Histopathological examination at low magnification demonstrated preserved epidermal architecture with focal epidermal alterations and marked papillary dermal edema (Figure 3). Higher magnification revealed intraepidermal and subcorneal pustules predominantly composed of neutrophils without evidence of spongiform pustules of Kogoj or regular psoriasiform epidermal hyperplasia (Figure 4). The superficial dermis showed a neutrophil-rich perivascular inflammatory infiltrate consistent with an acute drug-related inflammatory reaction pattern (Figure 5). Clinicopathological correlation supported the diagnosis of acute generalized exanthematous pustulosis (AGEP).

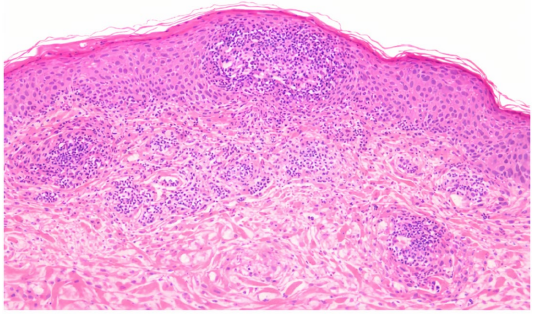

Figure 3. Low-power histopathologic view showing preserved epidermal architecture with focal epidermal alterations and prominent papillary dermal edema associated with a superficial inflammatory infiltrate.

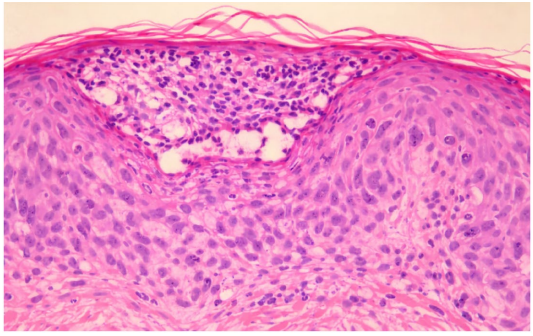

Figure 4. Higher-magnification view demonstrating intraepidermal and subcorneal pustule formation composed predominantly of neutrophils, without regular psoriasiform epidermal hyperplasia.

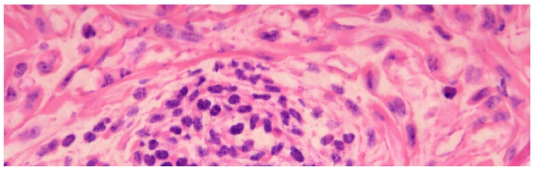

Figure 5. Superficial dermis showing a neutrophil-rich perivascular inflammatory infiltrate consistent with an acute drug-related inflammatory reaction pattern typical of AGEP.

Structured diagnostic assessment using validated AGEP criteria resulted in classification consistent with probable-to-definite AGEP, based on acute pustular eruption, compatible histopathology, absence of infection, and rapid improvement following withdrawal of the suspected drug.

Based on temporal association and absence of alternative triggers, adalimumab was identified as the most likely causative agent and was immediately discontinued, while leflunomide therapy was maintained.

Given the extensive cutaneous involvement, systemic treatment with cyclosporine was initiated at a starting dose of 3 mg/kg/day, administered in two divided doses. Blood pressure and renal function were monitored weekly during treatment. Rapid clinical improvement was observed within the first week, with progressive resolution of pustules and erythema. Cyclosporine therapy was maintained for four weeks followed by gradual tapering over an additional two-week period, achieving complete clearance of the acute eruption without adverse events.

Approximately six weeks after complete resolution of AGEP, the patient developed new cutaneous lesions with morphology distinct from the initial presentation. These consisted of well-demarcated erythematous scaly plaques with psoriasiform features predominantly involving the scalp and plantar regions. The delayed appearance after AGEP resolution suggested a paradoxical psoriatic reaction associated with prior anti-TNF-$\alpha$ exposure rather than persistence or relapse of pustular disease. Due to typical clinical morphology and stable course, repeat biopsy was considered but deferred.

During longitudinal follow-up, no recurrence of generalized pustulation occurred after adalimumab withdrawal. The patient remained under combined dermatologic and rheumatologic surveillance with sustained control of systemic disease.

Written informed consent for publication, including clinical and histopathological images (**Figures 1-5**), was obtained from the patient. According to institutional policy, formal ethics committee approval was not required for single anonymized case reports.

## 3. Discussion

Acute generalized exanthematous pustulosis (AGEP) is a prototypical severe cutaneous adverse reaction (SCAR) characterized by the abrupt onset of sterile, non-follicular pustules on an erythematous background, frequently accompanied by fever and neutrophilia, and typically showing rapid resolution after withdrawal of the causative agent [9]. Although AGEP has classically been associated with antibiotics, contemporary reviews and expert recommendations emphasize its increasing recognition in association with non-antibiotic drugs and modern immunomodulatory therapies, including biologic agents [10]-[12]. While the overall prognosis is usually favorable, systemic involvement occurs in a clinically relevant minority of cases, and severe presentations have been reported, reinforcing the importance of early recognition and appropriate classification [12].

This case is particularly instructive because it illustrates a clinically relevant interface between two biologic-associated inflammatory patterns: drug-induced AGEP and anti-tumor necrosis factor-$\alpha$ (TNF-$\alpha$)-associated paradoxical psoriasiform disease.

Anti-TNF agents are well recognized to induce paradoxical cutaneous inflammation, including de novo psoriasis and pustular variants, despite being therapeutically effective in psoriasis itself [13]. This paradox has been consistently documented across rheumatologic and gastroenterologic indications and may arise months after therapy initiation, supporting a mechanism of immune deviation rather than a classic immediate hypersensitivity reaction [14]-[16].

From an immunopathogenic standpoint, several complementary mechanisms have been proposed to explain why TNF-$\alpha$ blockade predisposes to pustular phenotypes.

Inhibition of TNF-$\alpha$ signaling may disinhibit plasmacytoid dendritic cell activity and enhance interferon-$\alpha$-driven pathways, promoting neutrophil-rich inflammation and epidermal immune activation [17]. These mechanisms conceptually align with both the acute neutrophilic pustulation characteristic of AGEP and the psoriasiform remodeling observed in paradoxical psoriasis. Importantly, this overlap does not imply diagnostic equivalence but rather suggests that a shared upstream immune perturbation may yield distinct downstream phenotypes depending on host susceptibility, timing, and the evolving inflammatory milieu.

The diagnostic strength of the present report rests on the clinicopathological coherence of the initial eruption. Clinically, the abrupt onset of widespread sterile-appearing pustules with prominent intertriginous clustering (**Figure 1**) and palmoplantar involvement (**Figure 2**) is highly compatible with AGEP, and the absence of a prior history of psoriasis lowers—though does not eliminate—the pre-test probability of pustular psoriasis. Histopathologic examination demonstrated features supportive of AGEP, including intraepidermal and subcorneal pustulation (**Figure 4**), papillary dermal edema (**Figure 3**), and a superficial perivascular infiltrate rich in neutrophils (**Figure 5**), without the regular psoriasiform epidermal hyperplasia or architectural features expected in chronic plaque psoriasis (17). These findings are concordant with canonical AGEP histology as defined in validated diagnostic frameworks. Nevertheless, the subsequent development of psoriasiform plaques involving the scalp and plantar regions following resolution of the acute pustular phase introduces the central diagnostic challenge of this case. In patients receiving biologic therapy, sequential phenotypes may occur, whereby an acute pustular drug eruption resolves and is followed by the emergence of psoriasiform disease. Clinically, this distinction is critical, as management strategies diverge substantially: AGEP is expected to remit after drug withdrawal, whereas paradoxical psoriasis may persist or recur and often necessitates ongoing topical or systemic therapy, as well as reconsideration of the biologic class used for the underlying inflammatory condition.

To address this complexity, we incorporated a pragmatic comparative framework (Table 1) emphasizing features most useful in routine clinical and histopathological practice, including timing of onset, presence of systemic symptoms and laboratory abnormalities, histopathologic architecture, and—most discriminating in clinical care—the evolution of the eruption after withdrawal of the suspected offending agent. In the present case, rapid resolution of the acute pustular eruption after discontinuation of adalimumab supported AGEP, whereas the later development of psoriasiform plaques favored a paradoxical anti-TNF phenomenon rather than persistent AGEP [18].

Table 1. Key clinical and histopathological features distinguishing AGEP from anti-TNF-induced paradoxical psoriasis.

| Feature | AGEP | Paradoxical psoriasis (anti-TNF) |
|---|---|---|
| Onset | Abrupt, acute | Subacute or delayed |
| Trigger | Drug exposure | Anti-TNF therapy |
| Pustules | Numerous, sterile, non-follicular | May be pustular or plaque-based |
| Distribution | Generalized, flexural, palmoplantar | Scalp, palms/soles, extensor surfaces |
| Systemic symptoms | Common (fever, malaise) | Usually absent |
| Laboratory findings | Neutrophilia, ↑ CRP (may be absent) | Usually normal |
| Histopathology | Subcorneal/intraepidermal pustules, dermal edema | Psoriasiform hyperplasia, spongiform pustules |
| Course after drug withdrawal | Rapid resolution | May persist or progress |

Management of extensive AGEP remains heterogeneous. While withdrawal of the causative agent and supportive care are foundational, systemic therapy is frequently considered in severe or extensive cases. Cyclosporine has emerged as a reasonable option in selected patients and has been supported by retrospective analyses as a potentially rapid and steroid-sparing intervention, particularly when disease burden is high or systemic corticosteroids are undesirable.

Finally, this case highlights important implications for interdisciplinary care. Because paradoxical psoriasis during anti-TNF therapy has been reported across multiple inflammatory disease populations, close collaboration between dermatology and rheumatology is essential when determining whether to re-challenge with the same agent, switch within class, or transition to an alternative biologic pathway. Moreover, when sequential phenotypes occur, longitudinal follow-up becomes diagnostically decisive, as the temporal behavior of the eruption often clarifies the distinction between AGEP and paradoxical psoriasis more reliably than any single clinical or histologic snapshot.

In summary, this report documents adalimumab-associated AGEP with subsequent psoriasiform evolution, reinforcing that biologic exposure may result in overlapping or sequential pustular and psoriasiform phenotypes. The case underscores the central role of clinicopathological correlation (Figures 3-5), the utility

of structured differentiation tools (Table 1), and the importance of longitudinal, multidisciplinary management to optimize outcomes in biologic-treated patients presenting with pustular eruptions.

Comparative summary of the main clinical, laboratory, and histopathological features useful for differentiating acute generalized exanthematous pustulosis (AGEP) from paradoxical psoriasis associated with anti-tumor necrosis factor (TNF) therapy. The table highlights differences in onset, triggers, pustule characteristics, systemic involvement, histopathologic architecture, and disease course after drug withdrawal.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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