



**GENERALIZED EXFOLIATIVE DERMATITIS ASSOCIATED WITH
CO-AMOXICLAV AND STAGE 2 PRESSURE ULCER**

Miranty Sasmita¹, Dina Arwina Dalimunthe^{1,2*}, Kristo Alberto Nababan^{1,3}

¹Department of Dermatology and Venereology, Faculty of Medicine, Universitas Sumatera Utara, Jl. Dr. Mansyur No.5, Padang Bulan, Medan Baru, Medan, Sumatera Utara 20155, Indonesia

²Prof. dr. Chairuddin P. Lubis, Universitas Sumatera Utara Hospital, Universitas Sumatera Utara, Jl. Dr. Mansyur No. 66, Merdeka, Medan Baru, Medan, Sumatera Utara 20154, Indonesia

³Adam Malik Hospital, Jl. Bunga Lau No.17, Kemenangan Tani, Medan Tuntungan, Medan, Sumatera Utara 20136, Indonesia

*dinadalimunthe15@gmail.com

ABSTRACT

Generalized exfoliative dermatitis (GED), or erythroderma, is a severe inflammatory skin disorder characterized by diffuse erythema and scaling involving more than 90% of the body surface area. Drug hypersensitivity is a common cause of GED, while extensive skin barrier disruption and systemic physiological disturbances may increase susceptibility to pressure ulcer development. Early pressure ulcer development in GED, particularly without prolonged immobilization, remains insufficiently recognized. This case is clinically significant because the pressure ulcer developed despite the absence of prolonged immobilization, suggesting that severe skin barrier dysfunction and systemic physiological disturbances may contribute to reduced tissue tolerance and early pressure-related tissue injury. This case report aims to highlight the clinical presentation, diagnostic challenges, and therapeutic management of drug-induced GED complicated by an early-onset Stage 2 pressure ulcer and emphasize the importance of early pressure ulcer risk assessment. We reported a 43-year-old woman who developed generalized erythema and diffuse scaling two days after taking co-amoxiclav and was diagnosed with drug-induced GED. Dermoscopy showed a diffuse erythematous background with superficial white scaling. Laboratory evaluation revealed a hemoglobin level of 10.8 g/dL. No pressure ulcer was present at admission; however, a Stage 2 pressure ulcer developed in the gluteal region on the third day. Data were obtained through anamnesis, physical and dermatological examination, dermoscopic evaluation, laboratory investigations, clinical observation, treatment interventions, and follow-up during hospitalization at Adam Malik Hospital, Medan. The patient received intravenous methylprednisolone 62.5 mg/day and topical desoximetasone 0.25% combined with urea and salicylic acid twice daily. The pressure ulcer was treated with topical gentamicin 1% cream twice daily, repositioning, and pressure offloading. Significant clinical improvement was observed by the ninth day. This case highlights the potential for early pressure ulcer development in drug-induced GED without prolonged immobilization, and underscores the importance of early risk assessment, pressure redistribution, repositioning, and preventive skin care.

Keywords: co-amoxiclav; drug hypersensitivity; erythroderma; generalized exfoliative dermatitis; pressure ulcer

How to cite (in APA style)

Sasmita, M., Dalimunthe, D. A., & Alberto Nababan, K. (2026). Generalized Exfoliative Dermatitis Associated with Co-Amoxiclav and Stage 2 Pressure Ulcer. *Indonesian Journal of Global Health Research*, 8(5), 1057–1066. <https://doi.org/10.37287/ijghr.v8i5.2474>.

INTRODUCTION

Generalized exfoliative dermatitis (GED), also known as erythroderma, is a rare but potentially life-threatening inflammatory dermatosis characterized by diffuse erythema and scaling involving more than 90% of the body surface area (Pang et al., 2024). The estimated incidence is approximately 1 per 100,000 adults, with erythroderma typically occurring between 41 and 61 years of age (Albalawi et al., 2024). It represents a clinical syndrome that may result from inflammatory dermatoses, malignancies, infections, drug reactions, or idiopathic causes (Pang et al., 2024). Drug-induced erythroderma may develop rapidly following medication exposure and can occur in individuals without a previous history of chronic dermatological disease (Morna et al., 2022; Pang et al., 2024). Drug-related cases account for approximately 20% of erythroderma cases, with β -lactam antibiotics, including co-amoxiclav, among the recognized triggers (Morna et al., 2022; Pang

et al., 2024; Zhu et al., 2021). The pathophysiology of GED involves increased production of pro-inflammatory cytokines and accelerated epidermal turnover, resulting in widespread epidermal barrier dysfunction, excessive transepidermal water loss, impaired thermoregulation, and systemic disturbances (Kliniec et al., 2024; Pang et al., 2024). Severe cases may be complicated by dehydration, electrolyte abnormalities, anemia, nutritional compromise, and secondary infection, potentially requiring hospitalization and close monitoring (Kliniec et al., 2024; Pang et al., 2024). Prompt withdrawal of the suspected offending drug and appropriate supportive management are essential, while systemic corticosteroids or other immunomodulatory therapies may be considered in selected patients (Jaruvijitrattana et al., 2026; Kliniec et al., 2024; Misturiansyah et al., 2024; Pang et al., 2024).

Systemic complications of erythroderma are well recognized, whereas the potential relationship between extensive skin barrier dysfunction and early pressure ulcer development has received limited attention. Pressure ulcer development is a multifactorial biomechanical process influenced by mechanical loading, pressure duration, shear, tissue deformation, perfusion, microclimate, and intrinsic tissue tolerance (Gefen et al., 2022; Portoghese et al., 2024). Severe GED may increase susceptibility to pressure-related tissue damage through the combined effects of epidermal barrier disruption, systemic inflammation, altered hydration, nutritional compromise, and reduced tissue resilience (Gefen et al., 2022; Velozo et al., 2025). These factors may become clinically relevant even during relatively short periods of reduced mobility or mechanical loading, particularly over pressure-prone areas such as the sacral and gluteal regions. The relationship between GED and early pressure ulcer development remains insufficiently characterized, and direct evidence that erythroderma independently increases pressure ulcer risk is limited. The potential interaction between skin barrier dysfunction, systemic physiological disturbances, and reduced tissue tolerance provides a plausible basis for early pressure ulcer development.

This case report aims to highlight the clinical presentation and diagnostic challenges of drug-induced GED following suspected co-amoxiclav use, complicated by an early-onset Stage 2 pressure ulcer, while describing the clinical features, diagnostic process, and therapeutic management of both conditions. It further aims to discuss the potential interaction between severe skin barrier dysfunction, systemic physiological disturbances, and reduced tissue tolerance in the development of early pressure-related tissue injury. The case also emphasizes the importance of early pressure ulcer risk assessment and individualized preventive strategies in patients with GED, even in the absence of prolonged immobilization.

METHOD

This study is presented as a single case report. Data were obtained through patient anamnesis, direct clinical observation, physical and dermatological examination, laboratory investigations, dermoscopic evaluation, and clinical follow-up. Only one patient was followed up throughout hospitalization, as this represents a single case report. The patient was selected because of the development of an early-onset Stage 2 pressure ulcer during hospitalization for suspected drug-induced GED following co-amoxiclav use. Data collection included clinical photographs, hematological test results, and dermoscopic findings. The information was descriptively analyzed and presented narratively, with the clinical findings and outcomes discussed in relation to existing literature on GED, drug-induced erythroderma, and pressure ulcer.

RESULT

A 43-year-old woman presented to the Dermatology Clinic with with generalized erythema, pruritus, and scaling persisting for eight weeks. The lesions initially appeared on the arms and gradually progressed to involve almost the entire body surface. The symptoms began two days after taking co-amoxiclav for a common cold. There was no history of prior dermatological disease, drug allergy, or similar condition. On physical examination, the patient appeared moderately ill but was

fully conscious, with stable vital signs. Dermatological findings revealed generalized, well-demarcated erythematous macules with diffuse scaling involving more than 90% of the body surface area. No erosion, ulceration, or pressure ulcer was observed at the time of admission (Fig. 1).

Dermoscopic examination showed a diffuse erythematous background with superficial white scaling (Fig. 2). Laboratory findings demonstrated anemia (hemoglobin 10.8 g/dL) and mildly elevated serum creatinine (1.3 mg/dL), while other parameters were within normal limits. Based on clinical findings and drug exposure history, a diagnosis of drug-induced generalized exfoliative dermatitis was established. The patient was hospitalized and treated with intravenous Ringer's lactate at 20 drops/ minute, intravenous methylprednisolone 62.5 mg/day, ranitidine 25 mg every 12 hours, oral chlorpheniramine maleate 4 mg three times daily, and oral zinc 20 mg once daily. Topical therapy consisted of topical corticosteroids combined with emollients consisting of desoximetasone 0.25% combined with urea and salicylic acid, applied twice daily.



Figure 1. Initial presentation. Generalized, well-demarcated erythematous macules with diffuse scaling involving the face (A), abdomen (B), back (C), upper extremities (D), lower extremities (E), and gluteal region (F). No erosion, ulceration, or tissue loss was observed at presentation. (Documentation by MS, DAD and KAN)



Figure 2. Dermoscopic findings. Diffuse erythematous background with superficial white scaling, consistent with exfoliative dermatitis. (DermLite DL200 Hybrid, $\times 10$, polarized). (Documentation by MS, DAD and KAN)

On the third day of hospitalization, improvement in erythema and scaling was observed; however, a new lesion developed in the gluteal region. Dermatological examination showed decreased erythematous macules and reduced scaling over the body, along with a superficial erosion in the gluteal region consistent with a Stage 2 pressure ulcer (Fig. 3). The diagnosis was updated to drug-induced GED with Stage 2 pressure ulcer. Treatment was adjusted by reducing intravenous fluids to 15 drops/minute and tapering methylprednisolone to 32.5 mg/day. Topical gentamicin 1% cream was added twice daily for the ulcer. The patient was also instructed to perform regular repositioning and use cushioning support to reduce pressure. Findings on day 4 showed persistent erythema with post-inflammatory hyperpigmentation and fine scaling was noted, while the gluteal ulcer remained without progression (Fig. 4). The diagnosis remained unchanged, and treatment was continued. Pressure offloading measures were maintained.

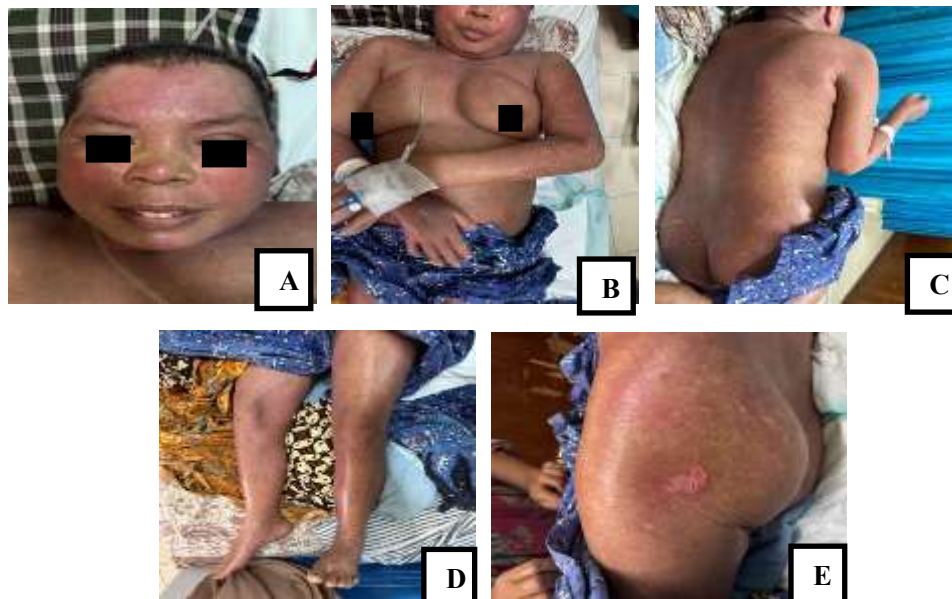


Figure 3. Day 3 of hospitalization. Improvement of erythematous macules with reduced scaling. A new superficial erosion consistent with a Stage 2 pressure ulcer is observed on the gluteal region. (Documentation by MS, DAD and KAN)



Figure 4. Day 4 of hospitalization. Persistent erythema with hyperpigmentation and fine scaling. The gluteal ulcer remains with no significant progression. (Documentation by MS, DAD and KAN)

By day 7, erythema and scaling had further improved, and the gluteal ulcer showed improvement with granulation tissue formation was observed (**Fig. 5**). The same treatment regimen was continued.



Figure 5. Day 7 of hospitalization. Further reduction of erythema and scaling. The gluteal ulcer shows signs of healing with granulation tissue formation. (Documentation by MS, DAD and KAN)

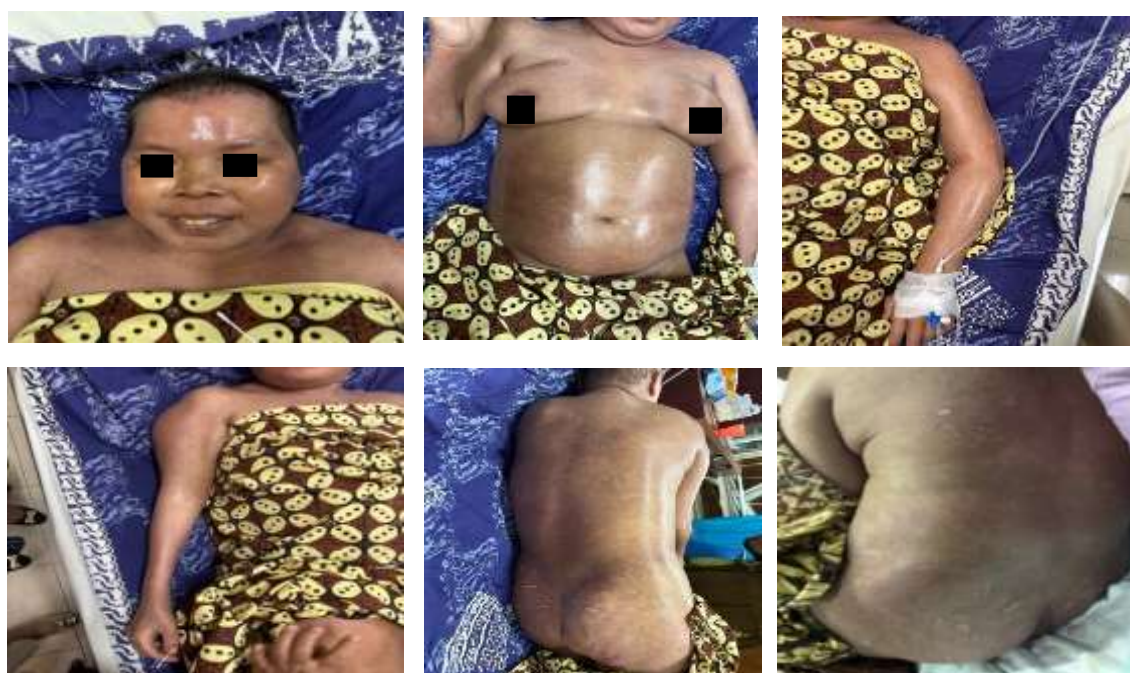


Figure 6. Day 9 of hospitalization. Predominantly hyperpigmented patches with minimal residual scaling. The gluteal lesion shows marked improvement with residual superficial erosion.

(Documentation by MS, DAD and KAN)

By day 9, both the erythroderma and ulcer showed significant improvement, leaving residual hyperpigmentation and minimal superficial erosion (Fig. 6). The final diagnosis remained drug-induced GED with Stage 2 decubitus ulcer. The patient was discharged in good condition with oral methylprednisolone 16 mg/day with gradual tapering, cetirizine 10 mg once daily, ranitidine 150 mg twice daily, vitamin B complex once daily, and topical therapies including hydrocortisone 1%

cream applied twice daily to the face, desoximetasone 0.25% combined with urea applied twice daily to the body, and gentamicin 1% cream applied twice daily to the gluteal region. The prognosis was considered good (*quo ad vitam, quo ad functionam, and quo ad sanationam*).

DISCUSSION

The development of a Stage 2 pressure ulcer on the third day of hospitalization in a patient with drug-induced GED represents a clinically important finding. The patient had no pressure ulcer at admission, yet a superficial lesion developed within less than 72 hours despite the absence of documented prolonged or complete immobilization. Pressure ulcer development is a multifactorial process determined by the interaction between external mechanical forces and the intrinsic capacity of tissues to tolerate and recover from mechanical stress (European Pressure Ulcer Advisory Panel et al., 2025; Gefen et al., 2022). The early development of the lesion in this case therefore suggests that factors beyond immobilization duration may have contributed to tissue breakdown. Severe GED is characterized by extensive cutaneous inflammation, accelerated epidermal turnover, and widespread disruption of epidermal barrier function. These abnormalities may result in increased transepidermal water loss, impaired thermoregulation, fluid and protein loss, and systemic physiological disturbances (Kliniec et al., 2024; Pang et al., 2024). Depending on disease severity and duration, patients may also develop dehydration, electrolyte abnormalities, anemia, nutritional compromise, and systemic inflammation. These manifestations are relevant to pressure ulcer development because tissue integrity depends not only on mechanical exposure but also on adequate perfusion, oxygenation, hydration, nutritional status, and the capacity for tissue repair. The coexistence of these factors reduce the ability of tissues to recover from repeated or sustained mechanical loading (Kliniec et al., 2024; Pang et al., 2024; Velozo et al., 2025).

The present case may therefore represent an interaction between disease-related tissue vulnerability and local mechanical exposure. In a patient with preserved tissue integrity and systemic homeostasis, intermittent mechanical loading may be compensated by spontaneous movement and physiological recovery. In contrast, when tissue tolerance is reduced, a similar degree of mechanical exposure may become clinically significant. This distinction is important because the absence of prolonged immobilization does not necessarily indicate a low risk of pressure ulcer in patients with severe GED, instead the patient's underlying disease may have reduced the threshold at which mechanical loading resulted in tissue injury. The skin barrier plays an important role in maintaining tissue integrity. The epidermis limits transepidermal water loss, protects against microbial invasion, and contributes to the maintenance of hydration and the local skin microenvironment. In GED, extensive erythema, scaling, and accelerated epidermal turnover may impair these functions across a large proportion of the body surface. Although GED does not typically cause the extensive epidermal detachment characteristic of Stevens–Johnson syndrome (SJS) or toxic epidermal necrolysis (TEN), the absence of full-thickness epidermal loss does not necessarily indicate preserved barrier function (Morna et al., 2022; Zhu et al., 2021). Widespread inflammation and desquamation may still compromise the functional integrity of the epidermis and reduce the resilience of superficial tissues.

The mechanism may be particularly relevant to the development of a Stage 2 pressure ulcer. The epidermis and superficial dermis are continuously exposed to friction, shear, moisture, and pressure during positioning and routine nursing care. When the skin barrier is compromised, these superficial layers may become more susceptible to mechanical disruption. Increased transepidermal water loss may contribute to altered hydration, while widespread inflammation may affect the structural and functional properties of the epidermis. These changes may not independently cause ulceration but may lower the amount of mechanical stress, that tissue can tolerate before breakdown occurs. GED may therefore function as a condition that reduces tissue resilience rather than as a direct cause of pressure ulcer formation. Pressure ulcer development involves pressure, shear, friction, tissue deformation, perfusion, and microclimate. Sustained mechanical loading over pressure-prone areas

can deform skin and underlying soft tissues, impair microvascular perfusion, obstruct lymphatic drainage, and reduce oxygen delivery. Prolonged tissue deformation may contribute to ischemia, impaired metabolic exchange, inflammatory responses, oxidative stress, and cellular injury (European Pressure Ulcer Advisory Panel et al., 2025; Gefen et al., 2022; Portoghese et al., 2024). Shear may further increase tissue strain by causing adjacent tissue layers to move in different directions, while friction may damage superficial epidermal structures. The resulting tissue damage depends on both the characteristics of the mechanical load and the intrinsic ability of the tissue to withstand it.

The concept of tissue tolerance provides a useful framework for understanding the present case. Tissue tolerance refers to the capacity of skin and underlying tissues to withstand mechanical loading without progressing to irreversible injury. This capacity varies between individuals and may be influenced by tissue composition, perfusion, nutritional status, systemic inflammation, age, hydration, skin condition, and microclimate (Gefen et al., 2022; Velozo et al., 2025). Patients with reduced tissue tolerance may develop pressure-related damage after shorter periods or lower intensities of mechanical loading than individuals with preserved tissue integrity. The development of a Stage 2 pressure ulcer within three days may therefore reflect a mismatch between the mechanical forces acting on the gluteal region and the reduced capacity of the affected tissues to tolerate those forces.

Tissue tolerance should also be considered a dynamic characteristic. Patients with severe GED may experience rapid changes in their clinical condition during hospitalization. Although mobility may initially be relatively preserved, fatigue, discomfort, pruritus, dehydration, thermoregulatory disturbances, or systemic illness may subsequently reduce spontaneous movement. At the same time, progressive fluid loss, nutritional compromise, anemia, or systemic inflammation may further reduce tissue tolerance. Consequently, both mechanical exposure and biological susceptibility may change over a short period. A patient who does not appear significantly immobile at admission may therefore become increasingly vulnerable to pressure ulcer development during the early course of hospitalization.

Several mechanisms associated with severe GED may contribute to reduced tissue tolerance. Extensive inflammation may alter local vascular function and increase metabolic demands within affected tissues. Increased transepidermal water loss may contribute to dehydration and changes in tissue hydration, while protein loss and nutritional disturbances may impair tissue repair and wound healing. Anemia may reduce oxygen delivery, and systemic inflammation may contribute to endothelial dysfunction and microcirculatory impairment (Kliniec et al., 2024; Pang et al., 2024; Velozo et al., 2025). These mechanisms may act simultaneously, creating a tissue environment that is less capable of recovering from repeated mechanical stress. In the present case, the acute inflammatory state associated with drug-induced GED may therefore have increased tissue vulnerability before the pressure ulcer became clinically apparent.

The location of the lesion in the gluteal region is also clinically relevant. The sacral and gluteal areas are recognized pressure-prone sites because they are exposed to mechanical loading during supine or semi-recumbent positioning (European Pressure Ulcer Advisory Panel et al., 2025). Local mechanical stress may be influenced by body weight, friction, shear, and repeated repositioning (Gefen et al., 2022; Portoghese et al., 2024). In patients with preserved tissue tolerance, intermittent loading may be compensated by spontaneous movement and microvascular recovery. In a patient with severe GED, however, reduced tissue tolerance may make the same anatomical region vulnerable despite relatively short periods of loading. The gluteal location may therefore represent the point at which systemic and local risk factors converged. The underlying GED may have reduced the physiological reserve of the skin and surrounding tissues, while the gluteal region was repeatedly exposed to mechanical loading. The lesion may consequently have

resulted from the interaction between a vulnerable tissue environment and a pressure-prone anatomical site. This interpretation is consistent with the multifactorial nature of pressure ulcer development and provides a plausible explanation for the early onset observed in this patient.

Friction and shear may have further contributed to the superficial nature of the lesion. Friction can damage superficial epidermal layers, particularly when the skin is inflamed, scaling, or otherwise compromised (Morna et al., 2022). Shear, in contrast, may cause deformation within deeper tissue layers and compromise local microvascular perfusion (European Pressure Ulcer Advisory Panel et al., 2025; Gefen et al., 2022). These forces may occur together during repositioning, patient transfers, or routine nursing care. In a patient with an already compromised epidermal barrier, relatively low-intensity mechanical forces may therefore contribute to superficial skin breakdown. This mechanism may partly explain why a Stage 2 lesion, rather than a deeper pressure ulcer, developed in the present case. Microclimate may represent an additional contributing factor. Temperature and moisture at the skin-support surface interface can influence skin condition and tissue response to mechanical loading (European Pressure Ulcer Advisory Panel et al., 2025). Excessive moisture may alter the mechanical properties of the stratum corneum, whereas increased temperature may increase metabolic demand and influence tissue susceptibility to pressure-related damage. Recent evidence indicates that heat and pressure exposure can affect erythema, skin temperature, and stratum corneum hydration in pressure-prone areas (Borzdynski et al., 2025). These findings may be particularly relevant to GED because impaired thermoregulation and extensive cutaneous inflammation may alter the local skin microenvironment. The combination of barrier dysfunction and an unfavorable microclimate may therefore further reduce tissue resilience.

The clinical morphology of the lesion provides an additional consideration. The lesion was consistent with a Stage 2 pressure ulcer, characterized by partial-thickness skin loss with exposed dermis and without exposed adipose tissue, granulation tissue, slough, or deeper structures (European Pressure Ulcer Advisory Panel et al., 2025). The superficial nature of the lesion may reflect the vulnerability of the epidermis and superficial dermis in the setting of widespread cutaneous inflammation. Pre-existing barrier dysfunction may have facilitated progression from erythema and superficial tissue stress to clinically evident partial-thickness skin loss when mechanical loading was sustained (Kandi et al., 2022). The development of a Stage 2 lesion may therefore be compatible with a predominantly superficial process occurring in skin that was already functionally compromised by GED (European Pressure Ulcer Advisory Panel et al., 2025). The temporal sequence of events provides further support for this interpretation. The patient had no pressure ulcer at admission, developed the lesion on the third day of hospitalization, and subsequently demonstrated improvement following treatment of GED and implementation of pressure-relieving measures. This temporal association does not establish that GED caused the pressure ulcer. It supports the hypothesis that the acute inflammatory and barrier-disrupting state associated with GED may have contributed to increased tissue susceptibility during hospitalization (Kliniec et al., 2024; Pang et al., 2024; Gefen et al., 2022). The absence of documented prolonged immobilization further suggests that intrinsic tissue vulnerability may have played an important role alongside local mechanical exposure (European Pressure Ulcer Advisory Panel et al., 2025; Gefen et al., 2022).

The subsequent clinical improvement is also consistent with a multifactorial process. Treatment of the underlying GED may have improved systemic physiological stability and skin barrier function, while pressure redistribution and off-loading reduced ongoing mechanical stress. The simultaneous management of these factors may have facilitated tissue recovery. However, the clinical response cannot be interpreted as proof that treatment of GED alone prevented further pressure ulcer progression. It supports the importance of addressing both the underlying disease and mechanical risk factors. Pressure ulcer prevention is particularly important in hospitalized patients with severe erythroderma. Risk assessment should be performed early, ideally at admission, and repeated when

the patient's clinical condition changes. Assessment should not rely solely on mobility or anticipated duration of immobilization but should also consider skin condition, systemic inflammation, hydration, nutritional status, anemia, perfusion, and other factors that may influence tissue tolerance (European Pressure Ulcer Advisory Panel et al., 2025; Mulianto et al., 2023). This approach is particularly relevant when the patient's condition is evolving rapidly.

Preventive strategies should include pressure redistribution, regular repositioning, appropriate support surfaces, frequent inspection of pressure-prone areas, and minimization of friction and shear during patient handling (European Pressure Ulcer Advisory Panel et al., 2025; Kandi et al., 2022; Mulianto et al., 2023). Skin care should be gentle and should avoid unnecessary mechanical trauma to inflamed and desquamating skin. Hydration and nutritional status should be assessed and optimized when abnormalities are present, while anemia and systemic physiological disturbances should be recognized and managed appropriately. Attention to skin microclimate may also be relevant, particularly in areas exposed to moisture, heat, or prolonged contact with support surfaces (Borzdynski et al., 2025). The timing of preventive interventions may be particularly important. Pressure ulcer prevention is often emphasized for patients expected to remain immobile for prolonged periods. The present case illustrates that this approach may overlook patients with substantial intrinsic tissue vulnerability who develop pressure-related injury within a short period. A single risk assessment at admission may therefore not accurately reflect the patient's risk during subsequent hospitalization. Changes in inflammation, hydration, nutrition, systemic stability, mobility, and skin condition may rapidly alter the balance between mechanical loading and tissue tolerance. Repeated assessment should consequently be integrated into the ongoing care of patients with severe GED, particularly when their clinical condition or skin integrity changes (Kandi et al., 2022).

The lesion developed within three days of hospitalization at a recognized pressure-prone site and presented as a superficial Stage 2 pressure ulcer. These findings support a multifactorial model in which severe GED may reduce tissue tolerance and interact with local mechanical forces to facilitate early pressure ulcer development (Kandi et al., 2022). The case does not establish that GED independently causes pressure ulceration; however, it suggests that conventional risk assessment based primarily on immobilization duration may underestimate pressure ulcer risk in patients with severe erythroderma. This case highlights the importance of early pressure ulcer risk assessment in patients with severe GED, even in the absence of prolonged immobilization. Preventive measures, including pressure redistribution, repositioning, appropriate skin care, microclimate management, and optimization of hydration and nutrition, should be initiated early and adjusted according to changes in clinical status (European Pressure Ulcer Advisory Panel et al., 2025; Kandi et al., 2022). Further studies are needed to clarify whether severe GED is independently associated with increased pressure ulcer risk and to identify predictors of early tissue breakdown.

CONCLUSION

This case highlights the early development of a Stage 2 pressure ulcer in a patient with drug-induced GED following suspected co-amoxiclav exposure, despite the absence of prolonged immobilization. Severe skin barrier dysfunction, systemic inflammation, and reduced tissue tolerance may contribute to early pressure ulcer development in patients with GED. Early risk assessment, pressure-relieving measures, and multidisciplinary supportive care should therefore be considered to prevent additional morbidity and improve clinical outcomes.

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