Localized Pustular and Plaque Psoriasis in a 27-Year-Old Woman: A Case Report

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Abstract. Psoriasis is a chronic immune-mediated inflammatory disease characterized by epidermal hyperproliferation, abnormal keratinocyte differentiation, and systemic inflammation. Psoriasis may present in several clinical forms, including plaque psoriasis and pustular psoriasis. This case report describes a 27-year-old woman who presented with multiple painful purulent lesions on both thighs, the abdomen, and the chest for one week, accompanied by fever, headache, and nausea. Dermatological examination revealed multiple purulent lesions with an erythematous base on both femoral regions, with desquamation on ruptured lesions. Multiple well-demarcated erythematous plaques with silvery-white scales were also observed on the trunk and dorsal region. Laboratory examination showed mild leukocytosis (WBC 12.44 $\times 10^3/\mu L$) and a random blood glucose of 284 mg/dL. The Psoriasis Area and Severity Index (PASI) score was 15.6, indicating moderate disease severity. The working diagnosis was localized pustular psoriasis associated with plaque psoriasis, with bullous pemphigoid and pityriasis lichenoides considered in the differential diagnosis. The patient received supportive and symptomatic treatment consisting of intravenous Ringer's lactate, paracetamol, omeprazole, dexamethasone, cetirizine, and carbamide cream. This case illustrates the importance of recognizing pustular lesions accompanied by systemic symptoms in patients with psoriasis and of objectively assessing disease severity using a validated scoring instrument. Appropriate management and monitoring are required, as pustular psoriasis may involve systemic complications and a recurrent disease course.

Keywords: Psoriasis, pustular psoriasis, plaque psoriasis, PASI.

1 Introduction

Psoriasis is a chronic, immune-mediated inflammatory skin disease associated with a range of systemic manifestations [1]. The disease is characterized by keratinocyte hyperproliferation, abnormal epidermal differentiation, and activation of multiple inflammatory pathways [2]. Clinically, psoriasis most commonly presents as psoriasis vulgaris, or plaque psoriasis, marked by well-demarcated erythematous plaques covered

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with silvery-white scales [1]. Other clinical variants include guttate psoriasis, inverse psoriasis, erythrodermic psoriasis, and pustular psoriasis [3].

Pustular psoriasis is a less common variant that can follow a more severe clinical course [5]. Its principal lesions are multiple sterile pustules that form as a result of neutrophilic infiltration of the epidermis [5]. In some cases, pustular psoriasis may be accompanied by systemic manifestations, such as fever, malaise, leukocytosis, and metabolic disturbances [6]. Because of these features, pustular psoriasis must be distinguished from other dermatoses that present with bullae, pustules, or papulosquamous lesions [8].

Psoriasis pathogenesis involves an interplay among genetic susceptibility, the immune system, and environmental factors [4]. Activation of dendritic cells and T cells drives the release of proinflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-17 (IL-17), IL-23, and IL-36 [5,8]. The IL-23/Th17 axis plays a central role in plaque psoriasis, whereas IL-36 has a more prominent role in pustular psoriasis through neutrophil activation and pustule formation [5,6]. Understanding these inflammatory pathways has underpinned the development of increasingly targeted psoriasis therapies [8].

Assessing disease severity is essential for guiding treatment selection and monitoring the disease course [9]. One of the most widely used instruments is the Psoriasis Area and Severity Index (PASI), which evaluates the extent of body surface involvement, degree of erythema, induration, and desquamation [9]. For patients with moderate-to-severe psoriasis, systemic therapy may be considered based on the patient's clinical condition and comorbidities [12,13].

This case report describes a 27-year-old woman with a clinical picture of plaque psoriasis, together with localized pustular psoriasis. This case is of interest because pustular lesions on the extremities were accompanied by silvery-white scaly plaques on the trunk, together with systemic symptoms, such as fever, headache, and nausea.

2 Case Report

A 27-year-old woman presented to the hospital on May 4 2026,, 2026 with painful, erythematous-based pustular lesions on both thighs, abdomen, and chest that had been present for one week before admission. The complaints had progressively worsened up to the day of examination. Several lesions had ruptured, leaving dried purulent crusts on an erythematous base. The cutaneous complaints were accompanied by fever, headache, and nausea. The patient had no relevant prior medical history and denied a history of hypertension or diabetes mellitus. Before admission, she had taken medication obtained from a local clinic. The family history was unknown, and the patient denied any known allergy to drugs, food, or other substances.

On physical examination, the patient appeared moderately ill, was conscious and oriented (GCS 15: E4V5M6), and had no focal neurological deficits. The blood pressure was 130/75 mmHg, pulse rate 87 beats/minute, respiratory rate 20 breaths/minute, temperature 38.0°C, and oxygen saturation 98%. General examination revealed no

edema, cyanosis, jaundice, or lymphadenopathy, and examination of the thorax, heart, abdomen, and extremities revealed no other significant abnormalities.

Dermatological examination of the bilateral femoral regions revealed multiple purulent bullae of varying sizes, the largest exceeding 1 cm in diameter, nummular in shape, well-demarcated, containing purulent (pustular) fluid clinically consistent with sterile pustules, although microbiological culture was not performed to formally confirm sterility, and set on an erythematous base; several ruptured lesions showed desquamation with an erythematous base. On the trunk, multiple scaly lesions were noted in the thoraco-abdominal area with an erythematous base, and on the dorsal region, multiple well-demarcated erythematous plaques of varying size and thickness with silvery-white scales were observed (Figure 1).



Fig. 1. Clinical images of femoral lesions showing purulent bullae with an erythematous base

Laboratory examination showed a hemoglobin level of 13.4 g/dL, an erythrocyte count of 6.32 $\times 10^6/\mu L$, hematocrit of 44.6%, MCV of 70.6 fL, MCH of 21.2 pg, MCHC of 30.0 g/dL, a leukocyte count of 12.44 $\times 10^3/\mu L$, and a platelet count of 338 $\times 10^3/\mu L$. The leukocyte differential count showed 0% basophils, 2% eosinophils, 58% segmented neutrophils, 34% lymphocytes, and 5% monocytes. Clinical chemistry showed a random blood glucose level of 284 mg/dL (Table 1).

Table 1. Laboratory findings.

Examination	Result	Reference range
Hemoglobin	13.4 g/dL	13–18 g/dL
Erythrocytes	6.32 $\times 10^6/\mu L$	3.8–6.2 $\times 10^6/\mu L$
Hematocrit	44.6%	39–51%
MCV	70.6 fL	79–99 fL
MCH	21.2 pg	27–31 pg

MCHC	30.0 g/dL	32–36 g/dL
Leukocytes	12.44 ×10³/µL	3.6–11 ×10³/µL
Platelets	338 ×10³/µL	150–440 ×10³/µL
Random blood glucose	284 mg/dL	<200 mg/dL

The patient's PASI score was 15.6, categorized as moderate-severity psoriasis [9]. Based on the history, physical examination, dermatological findings, and supporting investigations, the working diagnosis was localized psoriasis vulgaris et pustulosa. Bullous pemphigoid and pityriasis lichenoides were considered in the differential diagnosis [10,11]. The diagnosis in this case was established on clinical grounds, based on the history, physical examination, dermatological findings, and routine laboratory investigations; skin biopsy for histopathological examination and microbiological culture of the pustular lesions were not performed and were therefore not available to further confirm the diagnosis or to formally exclude other pustular or bullous dermatoses.

The patient received intravenous Ringer's lactate at 20 drops/minute, paracetamol 1000 mg every 8 hours by drip infusion, omeprazole 40 mg every 12 hours, dexamethasone 1 ampoule as adjunctive therapy, cetirizine 10 mg once daily, and carbamide cream twice daily [12,14,15]. The prognosis was assessed as *dubia ad bonam for quo ad vitam*, *quo ad functionam* and *quo ad sanationam*. These medications were administered during hospitalization; the clinical documentation available for this report did not record the exact total duration of inpatient treatment or any post-discharge follow-up assessment.

3 Discussion

This case illustrates a clinical presentation consistent with plaque psoriasis accompanied by localized pustular psoriasis. The patient was a young adult who presented with painful purulent lesions and fever. Such a presentation warrants particular attention, as pustular psoriasis can produce more pronounced systemic manifestations than ordinary plaque psoriasis [6,8]. In this patient, fever, headache, and nausea appeared concurrently with worsening of the cutaneous lesions.

Dermatological findings were the principal basis for the diagnosis in this case. Multiple purulent lesions with an erythematous base were found on both thighs, whereas well-demarcated erythematous plaques with silvery-white scales were found on the trunk and dorsal region. Silvery-white scaly plaques are a characteristic feature of plaque psoriasis [1,4], whereas pustular lesions on an erythematous base accompanied by systemic symptoms support the presence of a pustular psoriasis component [5,8]. Together, these two patterns formed the basis for the diagnosis of localized psoriasis vulgaris et pustulosa in this patient.

The clinical classification of pustular psoriasis in this case also warrants clarification. According to the European consensus statement on phenotypes of pustular psoriasis, pustular psoriasis is classified according to the distribution and extent of pustulation, ranging from localized forms to generalized pustular psoriasis (von Zumbusch type), which is characterized by widespread, rapidly progressive pustulation, typically accompanied by high-grade fever and marked systemic toxicity [18]. In this patient, the

pustular lesions were confined to the bilateral femoral regions rather than being widespread over the trunk and extremities, and the accompanying systemic features (low-grade fever, headache, and nausea) were comparatively mild rather than the marked systemic toxicity characteristic of generalized pustular psoriasis. This distribution and clinical severity support the classification of a localized pustular eruption superimposed on plaque psoriasis, rather than generalized pustular psoriasis; however, close monitoring remains warranted, as localized pustular psoriasis may evolve into a more generalized form [6,18].

Pathogenetically, psoriasis is an inflammatory disease involving the interaction between the innate and adaptive immune systems [4]. In pustular psoriasis, activation of the IL-36 pathway drives inflammation and neutrophil activation, leading to pustule formation, whereas the IL-23/Th17 pathway also helps sustain the inflammatory process [5,6]. Activation of multiple cytokines, including IL-17A, IL-23, TNF-α, and IL-36, is associated with keratinocyte proliferation, inflammatory cell infiltration, and cutaneous lesion formation [8,13].

The fever observed in this patient may have been related to the systemic inflammatory process accompanying pustular psoriasis. Pustular psoriasis can involve fever, malaise, leukocytosis, and metabolic disturbances [6,8]. In this case, the body temperature was 38.0°C, and the leukocyte count was 12.44 $\times 10^3/\mu L$, findings consistent with a systemic inflammatory response, although inflammatory biomarkers or other confirmatory investigations were not performed.

In addition to leukocytosis, a random blood glucose level of 284 mg/dL was found. This finding may be discussed in relation to the association between psoriasis and metabolic disturbance, including insulin resistance [12]. Psoriasis is a chronic inflammatory disease associated with metabolic syndrome and diabetes mellitus [12]. However, this patient had no prior history of diabetes mellitus, and further confirmatory testing for hyperglycemia was unavailable; therefore, the elevated random blood glucose should not be interpreted as diabetes mellitus without further evaluation. Beyond the recognized association between psoriasis and metabolic disturbance, the hyperglycemia in this patient may also have been partly influenced by the systemic inflammatory response and by dexamethasone, a glucocorticoid known to induce hyperglycemia through increased hepatic gluconeogenesis, reduced peripheral glucose uptake, and impaired insulin secretion [21]. Confirmatory investigations such as a repeat fasting or random plasma glucose measurement and glycated hemoglobin (HbA1c) were not performed in this case, so it was not possible to determine whether the hyperglycemia reflected an underlying, previously undiagnosed disturbance of glucose metabolism, a transient stress- and corticosteroid-related effect, or a combination of both. This gap highlights the importance of confirmatory glucose testing whenever hyperglycemia is identified in a hospitalized patient receiving systemic corticosteroids.

The PASI score was 15.6. PASI is an instrument used to assess psoriasis severity based on the extent of lesions together with the degree of erythema, induration, and desquamation [9]. This patient's score indicated moderate-severity disease according to the classification used. This assessment is important, as it can guide treatment selection and allow monitoring of clinical change during therapy [9]. The PASI is calculated by dividing the body surface into four regions (head, upper extremities, trunk, and lower

extremities), each scored for the severity of erythema, induration, and desquamation (0-4 for each parameter) and for the percentage of area involved (0-6), which are then combined using region-specific weighting coefficients to yield a total score ranging from 0 to 72 [9,20]. In this patient, the pustular and plaque lesions were distributed over the lower extremities (femoral regions), trunk, and dorsal region, and the total PASI score of 15.6 was derived from this regional scoring system. The individual regional and parameter-level scores underlying this total were not retained in the written clinical documentation available for this report and could therefore not be itemized in the manuscript; this is acknowledged as a limitation to the reproducibility of the PASI calculation, and future case reports should ideally present the full regional breakdown to allow independent verification.

The differential diagnoses considered in this case were bullous pemphigoid, pityriasis lichenoides, infectious pustular and bullous disorders, and drug-induced pustular eruptions, particularly given the history of medication use obtained from a local clinic before admission. Bullous pemphigoid typically presents with tense subepidermal bullae, whereas pityriasis lichenoides more commonly presents with papulosquamous lesions of a distinct character and distribution [10,11]. In this patient, purulent lesions on an erythematous base, together with plaques characteristic of psoriasis, favored a diagnosis of localized psoriasis vulgaris et pustulosa. Infectious causes such as bacterial folliculitis, furunculosis, or bullous impetigo may present with purulent, erythematous-based lesions and should be considered, especially without microbiological culture to exclude a bacterial etiology. Acute generalized exanthematous pustulosis (AGEP), a drug-induced pustular eruption characterized by the rapid onset of numerous small, non-follicular sterile pustules on an erythematous base together with fever and neutrophilic leukocytosis, is an important differential diagnosis whenever a pustular eruption follows recent drug exposure [19]. In this patient, the concurrent presence of classic psoriatic plaques with silvery-white scales favored a diagnosis of pustular psoriasis over AGEP; however, the precise identity, dose, and timing of the medication taken before admission were not available, and drug-induced pustulosis could therefore not be entirely excluded on the basis of the available clinical information.

Acute-phase management consisted of supportive and symptomatic therapy. Ringer's lactate was administered to help maintain fluid and electrolyte balance, particularly given the extensive cutaneous inflammation accompanied by fever. Paracetamol was given for fever and pain [14]. Omeprazole was used as adjunctive therapy during hospitalization, while cetirizine was given to help reduce discomfort and pruritus that may accompany psoriasis [15]. Ceramide cream was used as an emollient and keratolytic agent to improve skin hydration and reduce scaling.

Dexamethasone was also given as adjunctive therapy to suppress the acute inflammatory process. Systemic corticosteroids in psoriasis require caution, as abrupt discontinuation can precipitate a flare and, in certain circumstances, has been associated with transformation into more severe pustular psoriasis [6,8]. Systemic corticosteroids are therefore not recommended as long-term primary therapy for psoriasis, and their use should be undertaken cautiously and under close medical supervision. In this patient, dexamethasone was administered as a single ampoule (typically formulated as dexame-

thasone sodium phosphate, 5 mg/mL per ampoule in local clinical practice) given parenterally as adjunctive, short-term anti-inflammatory therapy over 3 days during the acute presentation, rather than as planned maintenance therapy. This limited, short-course use should be distinguished from the prolonged systemic corticosteroid regimens most strongly implicated in triggering rebound flares or conversion to generalized pustular psoriasis on withdrawal [6,8]; nonetheless, because this risk cannot be entirely excluded even after brief courses, tapering and close dermatological follow-up would have been advisable had the patient continued to be monitored after discharge. For moderate-to-severe pustular psoriasis, systemic therapy may be considered according to the patient's condition. Systemic treatment options include methotrexate, acitretin, cyclosporine, and biologic agents [13]. The development of biologic therapies has also provided more targeted options directed at inflammatory mediators such as TNF-α, IL-17, IL-23, and IL-36 [8,13]. Spesolimab, an anti-IL-36 receptor monoclonal antibody, has been described as one such targeted therapy used in generalized pustular psoriasis [6].

Regarding treatment outcomes, we had no data on clinical response, lesion resolution, change in PASI score, recurrence, adverse effects, or post-discharge management because we could not obtain follow-up after the initial hospitalization. This lack of outcome data limits our ability to evaluate the effectiveness of the management approach in this case. Had follow-up been available, clinically meaningful parameters would have included serial PASI scoring to document treatment response, monitoring for pustular recurrence or evolution toward a more generalized pattern, surveillance for corticosteroid-related adverse effects (including glycemic status), and confirmation of resolution of systemic symptoms; these parameters are recommended for future reporting of similar cases to allow more complete assessment of treatment outcomes [9,18].

Patient education is an important component of psoriasis management, given that the disease is chronic and prone to recurrence. Patients need to understand the importance of treatment adherence, skin care, and avoidance of precipitating factors such as stress, cutaneous trauma, infection, smoking, alcohol consumption, and abrupt discontinuation of medication [6]. Monitoring of metabolic status should also be considered in patients with psoriasis, particularly in those with elevated blood glucose findings, as observed in this case [12].

This case has several limitations. Microbiological and histopathological examinations were not reported, so no additional laboratory data were available to further support the diagnosis. Hormonal assessment and inflammatory biomarkers were likewise not performed. Complete follow-up data after treatment were also unavailable, so we could not assess the short-term treatment response or subsequent disease course. The elevated random blood glucose finding could not be classified as diabetes mellitus without further confirmatory testing. In addition, the documentation available for this report did not include itemized regional and parameter-level PASI scoring, limiting the reproducibility of the severity assessment and the granularity of the treatment description.

This case report has several limitations, including the absence of histopathological examination, microbiological testing, and inflammatory biomarkers that could have further supported the diagnosis and clarified the underlying pathogenesis. In addition,

complete follow-up data after treatment were unavailable, so we could not fully evaluate the treatment response and subsequent clinical course. The elevated random blood glucose level also requires further evaluation to determine whether an underlying glucose metabolism disorder is present. The detailed regional PASI component scores and the complete corticosteroid dosing and tapering record were likewise not available for inclusion in this report.

4 Conclusion

This case illustrates localized psoriasis vulgaris et pustulosa in a 27-year-old woman, manifesting as multiple purulent lesions with an erythematous base on the bilateral femoral regions together with erythematous, silvery-white scaly plaques on the trunk and dorsal region. Systemic complaints of fever, headache, and nausea, together with leukocytosis, supported the presence of an inflammatory response accompanying the pustular component. The PASI score was 15.6, consistent with moderate-severity psoriasis. The diagnosis was established based on the clinical presentation and available supporting investigations, with bullous pemphigoid and pityriasis lichenoides considered in the differential diagnosis. Initial management included supportive and symptomatic therapy, along with skin care. In moderate-to-severe pustular psoriasis, further evaluation and consideration of systemic therapy are required, depending on the patient's clinical condition.

Recognition of the pustular variant of psoriasis is important, as this presentation can be accompanied by systemic manifestations and carries a greater risk of complications than plaque psoriasis alone. Education regarding the chronic and recurrent nature of the disease, skin care, treatment adherence, avoidance of precipitating factors, and monitoring of comorbidities constitute important components of patient management.

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Disclosure of Interests. The authors declare no conflicts of interest.

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