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Severe Forms of Psoriasis: When Dermatological Management Meets Intensive Care Medicine

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Abstract **Case Report**

Background: Psoriasis is a chronic immune-mediated inflammatory disease that typically follows a controllable course under dermatological management. However, severe phenotypes may evolve into life-threatening systemic conditions requiring intensive care unit (ICU) admission. Despite this risk, psoriasis remains under-recognized as a cause of critical illness, and guidance on ICU management is limited. ***Objectives:*** To illustrate the spectrum of critical complications associated with severe psoriasis, identify clinical warning signs necessitating ICU admission, and highlight key principles of intensive care management through representative clinical cases. ***Methods:*** We report three severe and illustrative cases of psoriasis complicated by life-threatening systemic manifestations: methotrexate toxicity in end-stage renal disease, erythrodermic psoriasis, and generalized pustular psoriasis complicated by drug reaction with eosinophilia and systemic symptoms (DRESS). Clinical presentation, laboratory findings, therapeutic interventions, and outcomes are analyzed in the context of current literature. ***Results:*** All cases demonstrated rapid progression from cutaneous disease to systemic inflammatory failure. Two patients developed multiorgan dysfunction and died despite intensive care management. One patient with erythrodermic psoriasis recovered following aggressive ICU stabilization and subsequent biologic therapy. These cases underscore the role of systemic inflammation, immunosuppression, infection, and drug toxicity in driving critical illness among patients with psoriasis. ***Conclusions:*** Severe psoriasis should be regarded as a multisystem inflammatory disorder with the potential for critical deterioration. Early recognition, strict pharmacovigilance, prompt ICU referral, and close collaboration between dermatologists and intensivists are essential to improving outcomes. Integrated care pathways bridging dermatology and intensive care medicine are urgently needed for high-risk patients.

Keywords: Psoriasis; Intensive care; Erythrodermic psoriasis; Generalized pustular psoriasis; Methotrexate toxicity; DRESS syndrome; Critical illness; Multiorgan failure; Multidisciplinary management.

INTRODUCTION

Psoriasis is a chronic immune-mediated inflammatory skin disease affecting approximately 2–3% of the adult population worldwide [1]. In most cases, it follows a relapsing but manageable course, treated in outpatient settings with topical therapies, phototherapy, or systemic agents. However, a subset of patients may develop severe and potentially life-threatening forms. In these situations, psoriasis extends beyond the skin to become a systemic inflammatory disorder, capable of inducing hemodynamic instability, metabolic derangements, acute organ dysfunction, and overwhelming infection.

As a result, some patients with severe psoriasis require admission to intensive care units (ICUs) [2], where management priorities shift from disease control alone to life-supporting interventions.

Despite these risks, severe psoriasis remains under-recognized as a cause of critical illness. The literature addressing its management in the ICU is limited, fragmented, and often extrapolated from burn care or severe drug reaction protocols.

Through the presentation of three severe and illustrative cases, this article aims to emphasize the spectrum of critical complications associated with

psoriasis, ranging from treatment-related toxicity and erythrodermic collapse to drug-induced hypersensitivity syndromes. We seek to highlight the clinical warning signs necessitating ICU admission, discuss key principles of intensive care management in severe psoriasis, and underline the importance of multidisciplinary strategies to improve patient outcomes.

Case 1:

A 59-year-old patient undergoing hemodialysis three times per week for the past four years due to end-stage chronic kidney disease secondary to hepatorenal polycystic disease received a first injection of 10 mg methotrexate for the treatment of psoriasis extension.

Three days after drug administration, the patient developed a clinical picture characterized by pharyngitis, dysphagia, and odynophagia, for which amoxicillin-clavulanic acid was initiated. The condition progressed with the onset of cheilitis, stomatitis with inflammation of the pharyngeal mucosa, as well as multiple buccal and nasal erosions (Figure 1). Additionally, painful erythematous to violaceous skin lesions interspersed with pustules were observed on the back and palms, accompanied by desquamation (Figure 2). A tender erythematous plaque was also present on the external genital organs (Figure 3). These manifestations developed in a context of general health deterioration with fever reaching 38.8°C.

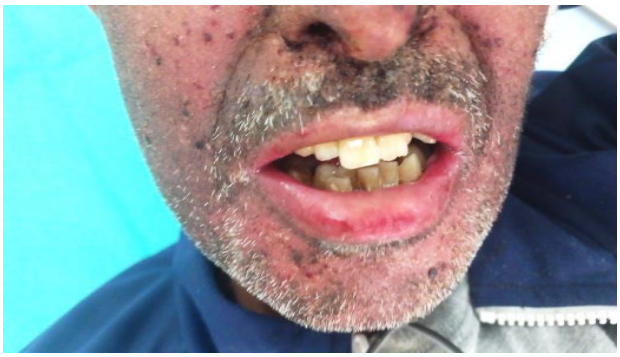

Figure 1: Cheilitis and erosions of the nasal cavities

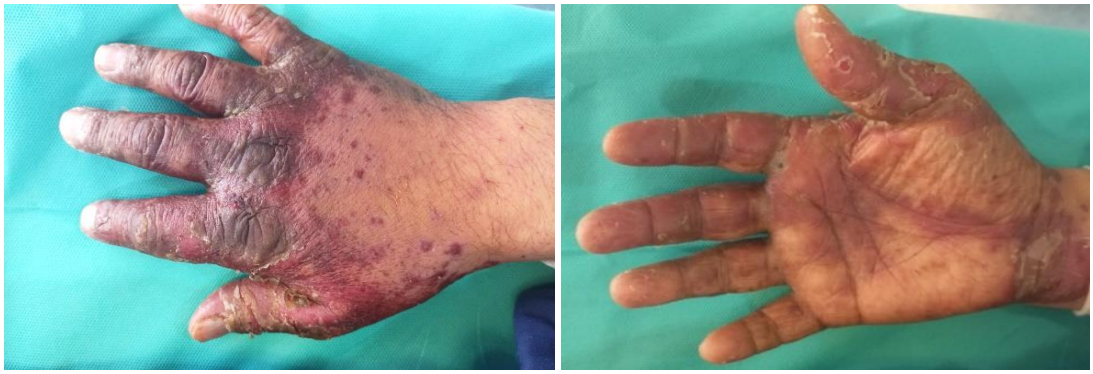

Figure 2: Acral erythema of the back and palms of the hands, evolving into desquamation

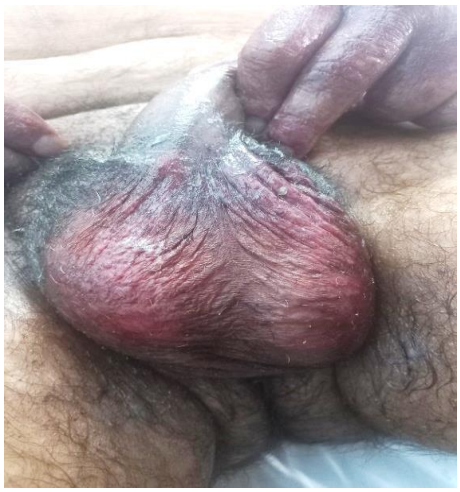

Figure 3: Erythème des organes génitaux externes

Histological examination of the skin lesions revealed epidermal detachment with basal layer necrosis, associated with spongiosis and exocytosis. The dermis was edematous with a perivascular inflammatory infiltrate and involvement of the dermo-epidermal junction, composed of lymphocytes and polymorphonuclear cells.

Biological workup revealed severe leukoneutropenia (leukocytes at 300/mm3), hemoglobin at 12.5 g/dL, and platelets at 163,000/mm3. A decrease in serum folate levels (2.8 ng/L) was noted, while the rest of the laboratory tests were unremarkable.

Given the severity of the condition, the patient was admitted to the intensive care unit for close monitoring and supportive management. Methotrexate was immediately discontinued, and treatment was initiated, including the administration of calcium folinate (100 mg, four injections), filgrastim (30 MUI, two injections), and skin care with neutral emollients and bicarbonate-based mouthwashes.

Despite intensive care measures, the patient's condition deteriorated, leading to the development of septic shock related to febrile neutropenia, ultimately resulting in death.

Case 2

A 64-year-old obese woman (elevated body mass index of 42), has been followed in the dermatology department since 1990 for psoriasis affecting large body areas. Initial treatment included topical corticosteroids, emollients, and phototherapy, but without significant clinical improvement.

In 2018, methotrexate was initiated at a dose of 25 mg per week, resulting in a favorable clinical response. However, due to the prolonged treatment, a FibroTest was performed and revealed signs of hepatic fibrosis, prompting discontinuation of methotrexate. In March 2023, the patient was hospitalized for a new flare of her disease.

On admission, she was conscious and breathing comfortably, with normal vital signs (Blood pressure of 120/70 mmHg). Dermatological examination revealed well-defined, pruritic, erythematous-scaly plaques involving the back, abdomen, and elbows, affecting approximately 40% of the body surface area (Figure 4). Her PASI score was 13 and DLQI was 10/30. The remainder of the physical examination was unremarkable.

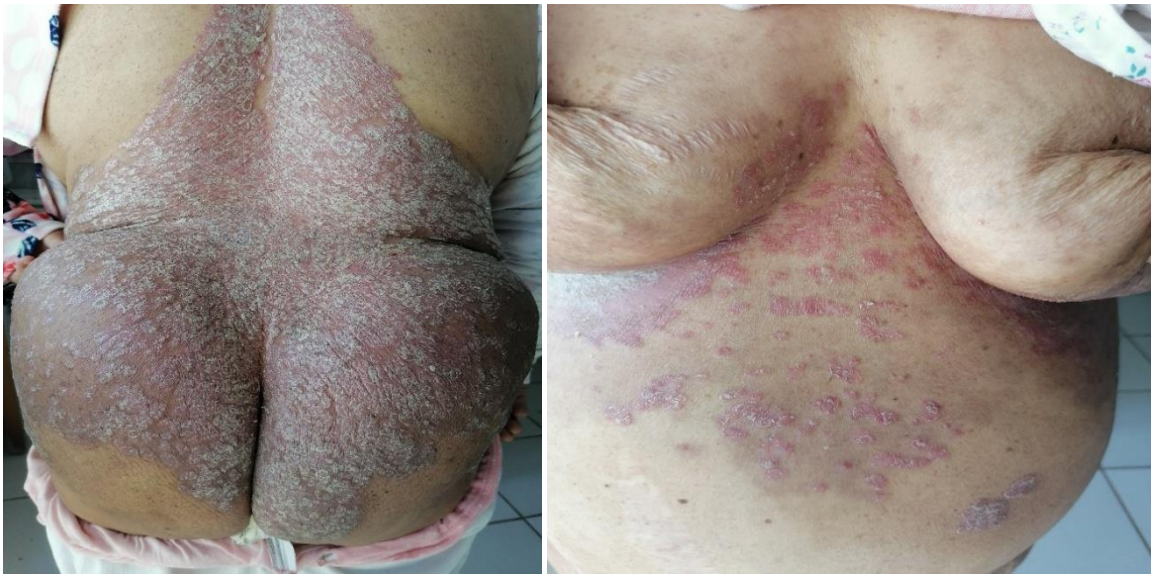

Figure 4: Extensive erythematous scaly lesions of the trunk

One week later, she developed generalized erythematous lesions covering 98% of her body surface area, accompanied by fever (39°C) and respiratory distress. A diagnosis of erythroderma was made (Figure 5).

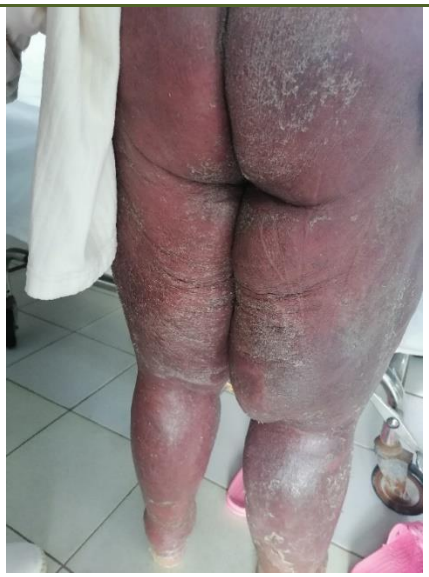

Figure 5: Wet psoriatic erythroderma

Her clinical course was complicated by the development of sepsis. On examination, she remained conscious, with elevated blood pressure (BP 160/90 mmHg), tachycardia (120 bpm), fever (38°C) and tachypnea.

Laboratory investigations showed a C-reactive protein (CRP) level of 283 mg/L, procalcitonin of 18 ng/L, leukocytosis (WBC 25,000/mm³), normocytic normochromic anemia and functional acute kidney injury (creatinine 43 mg/L, urea 0.73 g/L, hyperkalemia at 6.40 mmol/L). Given the severity of her condition, the patient was transferred to the intensive care unit, where she received the following treatment: voriconazole 200 mg every 12 hours, piperacillin-tazobactam 1 g/day, Colimycine 3 million IU/day, teicoplanin 130 mg/day, and omeprazole 40 mg/day. After 15 days in the ICU, the patient showed significant clinical and laboratory improvement.

She was subsequently transferred back to the dermatology department for ongoing care. One month later, treatment with ustekinumab at a dose of 90 mg was initiated, leading to complete clearance of skin lesions after the second dose (Figure 6).

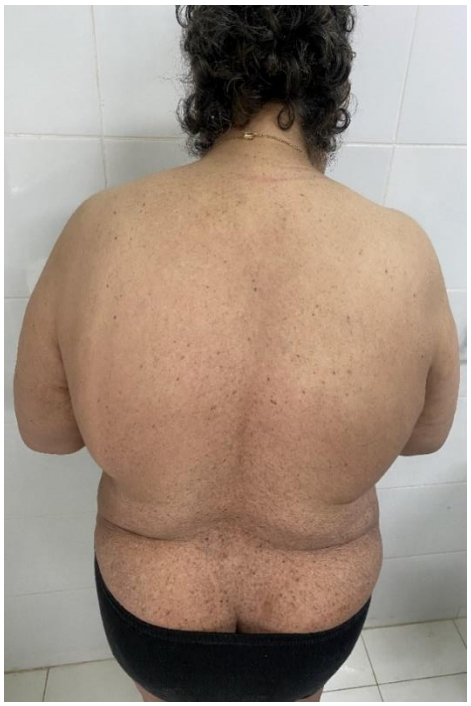

Figure 6: Complete clearing of psoriatic lesions after treatment with biotherapy

Case 3:

A 45-year-old man, with no significant past medical history, had been followed in dermatology for ten years for chronic plaque psoriasis. Over the course of the disease, he received several treatments adapted to the severity of his condition, including topical therapies (dermocorticoids, emollients, and keratolytics), UVB phototherapy (30 sessions, discontinued due to lack of efficacy), and systemic methotrexate (15 mg/week), which was stopped because of hepatotoxicity.

Approximately one month prior to admission, following a short course of oral corticosteroids (prednisone 60 mg/day), the patient developed a sudden-onset, generalized erythematous eruption characterized by annular plaques surmounted by peripheral, whitish pustules, predominantly involving the trunk, forearms, legs, and feet. The clinical picture was consistent with generalized pustular psoriasis (Figure 7).

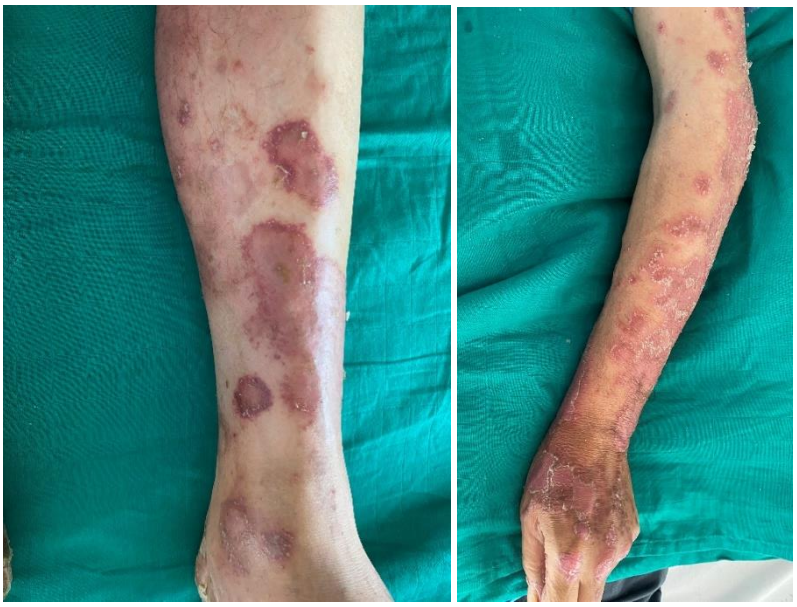

Figure 7: Erythematous lesions topped with pustules on the legs and forearms

Given the extent and severity of the lesions, initiation of biologic therapy (secukinumab) was considered. A comprehensive infectious screening was performed prior to treatment, revealing a positive Quantiferon-TB test. Consequently, anti-tuberculous therapy was started and maintained for one month before the planned initiation of the biologic agent.

After two weeks of anti-tuberculous therapy, the patient developed diffuse, pruritic erythematous eruptionsassociated with facial edema. A diagnosis of Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) syndrome secondary to the anti-tuberculous treatment was suspected and subsequently confirmed according to the Japanese consensus group criteria.

The anti-tuberculous regimen was immediately discontinued, and oral corticosteroid therapy (prednisone 1 mg/kg/day) was initiated. However, after one week of treatment, the patient's condition deteriorated, progressing to acute multiorgan dysfunction that required transfer to the intensive care unit. Despite appropriate management, the clinical course was rapidly unfavorable, and the patient ultimately died from systemic organ failure.

DISCUSSION

Severe forms of psoriasis represent a critical intersection between dermatology and intensive care medicine, where cutaneous disease transcends the limits of the skin and becomes a systemic, potentially life-threatening condition.

While psoriasis is most often managed in outpatient dermatology settings, the cases presented here illustrate how certain phenotypes—erythrodermic psoriasis, generalized pustular psoriasis, and severe treatment-related complications—can rapidly progress to conditions requiring intensive care medicine. These observations are supported by emerging epidemiological data demonstrating a substantial burden of ICU utilization among patients with psoriasis 3.

A population-based study by Marrie *et al.,* provides evidence that psoriasis is independently associated with an increased risk of critical illness. Using a large Canadian administrative health database, the authors demonstrated that patients with psoriasis had a significantly higher incidence of ICU admission compared with matched controls (21% higher), even after adjustment for age, sex, socioeconomic status, and comorbidities. Importantly, this increased risk was not limited to patients with severe or hospitalized psoriasis,

suggesting that psoriasis itself represents a systemic disease state predisposing to critical illness, rather than ICU admission being solely attributable to treatment-related complications or advanced disease severity.

Beyond ICU utilization, the study revealed a substantial excess mortality associated with psoriasis once critical illness occurs. Crude ICU mortality among patients with psoriasis was 11.5%, compared with 8.7% in matched ICU controls, corresponding to a 32% relative increase in ICU mortality. This mortality gap persisted well beyond hospital discharge, with higher mortality observed at 30 days, 1 year, and up to 5 years after ICU admission. Other studies confirm the same conclusion [4,5].

These findings underscore that psoriasis itself should not be viewed as benign in hospitalized patients, particularly when compounded by immunosuppression, renal dysfunction, or extensive skin involvement.

The pathophysiological continuum between skin and systemic inflammation explains why patients with extensive psoriasis are at higher risk for severe metabolic, infectious, and immunological complications.

At the core of psoriasis pathogenesis lies a complex cytokine network dominated by the IL-23/IL-17/TNF-α axis, involving a pathogenic triad of dendritic cells, Th17 cells, and hyperactivated keratinocytes [6]. This inflammatory loop is not confined to the skin. Elevated circulating levels of IL-17, IL-6, TNF-α, IL-22, and IL-1 family cytokines have been consistently demonstrated [6], providing a mechanistic explanation for the increased susceptibility of patients with psoriasis to systemic complications, including sepsis, cardiovascular instability, and multiorgan dysfunction. In this context, the threshold for intensive care admission should be low when clinical decompensation, fever, or metabolic imbalance occurs.

The first case highlights the catastrophic potential of methotrexate toxicity in patients with impaired renal clearance. Despite its efficacy, methotrexate requires meticulous dosing and strict contraindication assessment, especially in end-stage renal disease. The multicentric retrospective study by Singh *et al.,* provides one of the most comprehensive real-world evaluations of MTX toxicity in psoriasis, offering critical insights into its clinical spectrum, risk factors, and outcomes [7]. In their cohort of 21 patients hospitalized for MTX toxicity, over 95% exhibited mucocutaneous ulcerations and/or erosions, hematological toxicity was present in 76%, including leukopenia, thrombocytopenia, or pancytopenia, and three patients (14%) died due to sepsis and multiorgan failure These findings closely parallel the fatal evolution observed in our first case. In this patient, accumulation of the drug, compounded by folate depletion and infection, precipitated pancytopenia and mucocutaneous necrosis. The progression to septic shock despite early rescue therapy with folinic acid and granulocyte growth factor underscores the fragility of this population and the importance of preventive pharmacovigilance. Intensive care management in such scenarios should prioritize neutropenic sepsis protocols, early antimicrobial coverage, and organ support rather than dermatologic therapy alone.

The second case exemplifies erythrodermic psoriasis—a dramatic manifestation involving more than 90% of the body surface area. This condition disrupts thermoregulation, electrolyte balance, and skin barrier integrity, transforming the skin into a massive inflammatory organ. The resultant capillary leak, fluid losses, and increased metabolic demand frequently lead to hemodynamic instability and acute kidney injury. ICU management in such patients parallels that of extensive burns: active temperature control, correction of fluid and electrolyte imbalances, nutritional optimization, and prevention of secondary infections.

The patient's improvement after aggressive anti-infective therapy and subsequent transition to biologic treatment illustrates the benefit of bridging intensive care stabilization with targeted immunomodulation once the acute phase resolves [8]. This sequence highlights the critical collaboration between dermatologists and intensivists—where life-saving stabilization paves the way for long-term disease control.

The third case demonstrates another challenge: the overlap between severe psoriasis and drug-induced hypersensitivity syndromes. Generalized pustular psoriasis may mimic or coexist with severe drug reactions such as DRESS [9], both driven by immune overactivation. The triggering role of corticosteroid withdrawal [10] and subsequent exposure to anti-tuberculous agents [11] in this patient reveals the delicate immunological balance underlying psoriasis. Once systemic inflammation becomes uncontrolled, multiorgan failure can occur despite adequate intensive care measures. This fatal evolution emphasizes the need for early dermatologic–immunologic collaboration, cautious introduction of immunosuppressive or anti-infective drugs, and close biological surveillance.

For dermatologists, the ICU represents a domain where cutaneous signs often reflect systemic dysfunction, and where dermatologic emergencies—such as erythroderma, pustular flares, or toxic epidermal necrolysis-like reactions—require critical care logistics: thermal regulation, infection control, and organ support. Conversely, for intensivists, psoriasis crises serve as paradigms of immune-mediated systemic inflammation, illustrating how skin disease can become a systemic syndrome. This bidirectional learning reinforces the concept that severe psoriasis should be managed not only

as a dermatological entity but as a multisystem disorder necessitating integrated care pathways.

The fatal outcomes in two of the three cases underscore the urgency of prevention and early recognition. Pre-treatment screening for renal, hepatic, and infectious risk factors; proactive folate supplementation during methotrexate therapy; and early initiation of biologics in high-risk or refractory patients could prevent progression to critical illness. Furthermore, the transition between dermatology and intensive care must be fluid, with standardized referral criteria and shared treatment algorithms [12].

CONCLUSION

Severe psoriasis constitutes a bridge between dermatology and intensive care medicine—where inflammatory skin disease becomes a systemic emergency. Early multidisciplinary coordination, vigilant monitoring of drug toxicity, and timely escalation to biologic or targeted therapies are essential to improving outcomes. The presented cases highlight both the therapeutic challenges and the life-saving potential of close collaboration between dermatologists and intensivists in the management of complex inflammatory skin disorders.

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