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Erythrodermic Psoriasis: A Case Report

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ABSTRACT

Erythrodermic psoriasis is a rare, but serious subtype of psoriasis characterized by widespread erythema affecting over 75% of the total body surface area. It is a condition that may be mistaken for pathologies with similar clinical presentation, including seborrheic dermatitis or atopic dermatitis. A missed diagnosis of erythrodermic psoriasis may lead to life-threatening systemic complications including hypothermia, sepsis, and high-output heart failure. In this report, we introduce a case of erythrodermic psoriasis with acute progression after an initial diagnosis of new-onset atopic dermatitis, and highlight the clinical distinctions between the two pathologies.

Keywords: case report, erythrodermic psoriasis, atopic dermatitis

INTRODUCTION

Psoriasis is an immune-mediated overproduction of keratinocytes, resulting in widespread patches and plaques.[1] Erythrodermic psoriasis (EP) is a rare and severe subtype, affecting only 1-2.25% of psoriasis patients.[2] The clinical presentation of EP includes erythema of at least 75% of the total body surface area with peeling plaques of skin and widespread desquamation.[2] Chills, edema, fever, arthralgia, myalgia, lymphadenopathy, malaise, and fatigue are commonly associated symptoms.[3] If left untreated, EP may lead to systemic complications including hypothermia, sepsis, cachexia, and high-output heart failure secondary to significant water loss. This contrasts with the more common plaque psoriasis, characterized by well-defined pruritic plaques on the extensor surfaces with a characteristic, thickened overlaying silver scaliness and an absence of systemic symptoms.[1]

Risk factors for developing erythrodermic psoriasis include, but are not limited to, personal or family history of psoriasis, and various medical illnesses. For instance, while rare in the general population, EP is among the more common presentations of psoriasis in patients diagnosed with HIV.[4] Erythroderma psoriasis flares can also arise from uncontrolled disease, concomitant infection of Staphylococcus aureus or Streptococcus pyogenes, stress, or certain medications including acitretin, etretinate, lithium, antimalarials, and trimethoprim/sulfamethoxazole.[2,5] Withdrawal from previous psoriasis treatment is also associated with the development of EP.[5]

Treatment options vary depending on the severity of the case but largely consist of immunosuppressive medications. Guidelines published by the US National Psoriasis Foundation advocate for the use of cyclosporine and infliximab as first-line therapy in unstable cases requiring rapid response to treatment.[2,6] Some patients who do not respond well to the first-line therapies may require adalimumab or etanercept.[2,6] Recommended first-line therapies for stable patients not requiring emergent therapy include acitretin or methotrexate. Others require an oral or topical corticosteroid.[2,6] EP is frequently mistaken for other causes of erythroderma such as seborrheic dermatitis or atopic dermatitis, and as such these diagnoses must also be considered by clinicians.[2] In this article, we report a rare case of erythrodermic psoriasis with acute progression from an initial diagnosis of new-onset atopic dermatitis.

CASE PRESENTATION

An otherwise healthy 80-year-old male with a past medical history significant only for biopsy-proven atopic dermatitis presented to the emergency department due to persistent hypothermia (96 °F) and inability to maintain body temperature. The patient had been treating his recently diagnosed atopic dermatitis with intermittent use of topical triamcinolone. His total body surface area affected by the rash was estimated to be 90%. Furthermore, he presented with eruptive seborrheic

keratoses on his back, which had developed over the course of 1-2 weeks (Fig. 2A). Electrocardiogram demonstrated that the patient showed new-onset atrial fibrillation with rapid ventricular response, with a rate consistently tachycardic in the 120s. The cardiac MRI showed left ventricular wall hypertrophy with an ejection fraction of 40%, suggesting worsening heart function, though there was no clinical evidence of overt heart failure on presentation. Laboratory testing included elevated lactate dehydrogenase (279 U/L), and complete blood count was within normal limits with no atypical lymphocytes or Sézary cells observed on peripheral smear. Electrolyte imbalances are of major concern as significant water loss is possible during EP flares, however the patient's electrolytes were within normal limits. The remainder of his BMP was also normal, precluding any renal involvement. Considering erythroderma, atopic dermatitis, psoriasis, and Cutaneous T-cell Lymphoma/Mycosis Fungoides in the differential, a 4 mm punch biopsy was taken from the anterolateral aspect of the abdomen for histopathological review (Fig. 2B).

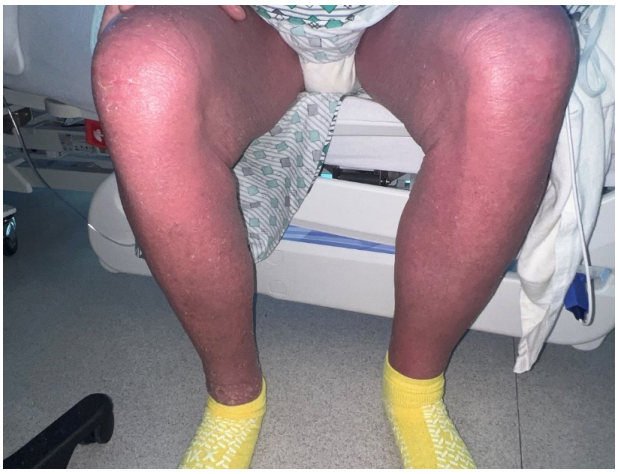

Figure 1. Widespread erythematous rash covering the anterior leg, with a peeling rash noted on the knees.

In preparation for histopathological analysis, the entirety of the formalin-fixed skin punch was submitted and serially sectioned after bisection for histopathologic examination. Sections revealed a skin biopsy extending to the deep reticular dermis (Fig. 3). Hyperkeratosis and nearly confluent regions of parakeratosis, with accumulations of clustered neutrophils were noted within the stratum corneum. This was found to overlie epidermis notable for acanthosis, spongiosis, and hypogranulosis, findings consistent with psoriasiform dermatitis.[7] Within the superficial dermis, there is a distinct perivascular chronic inflammatory infiltrate with relatively numerous scattered eosinophils and distinct capillary dilation. There is no evidence of either an interface reaction or of atypical lymphocytes.

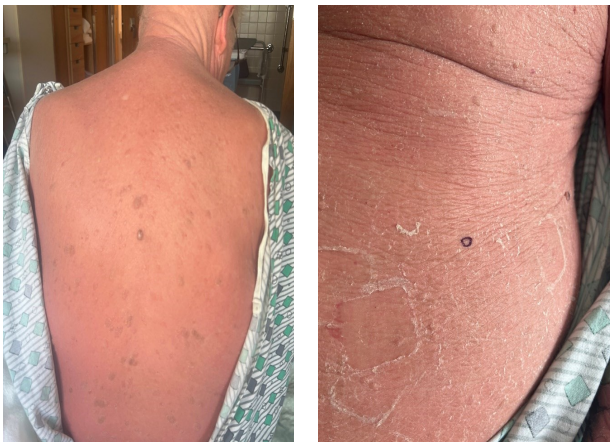

Figure 2. (A) Eruptive seborrheic keratosis discovered on patient's back at time of presentation. **(B)** Region of erythematous, scaling anterolateral abdomen from which a 4 mm punch biopsy was taken for a histopathological analysis.

The patient was discharged on a Prednisone taper as a bridging therapy and with an urgent appointment with a local dermatologist to discuss long-term psoriasis management with traditional first-line therapies.

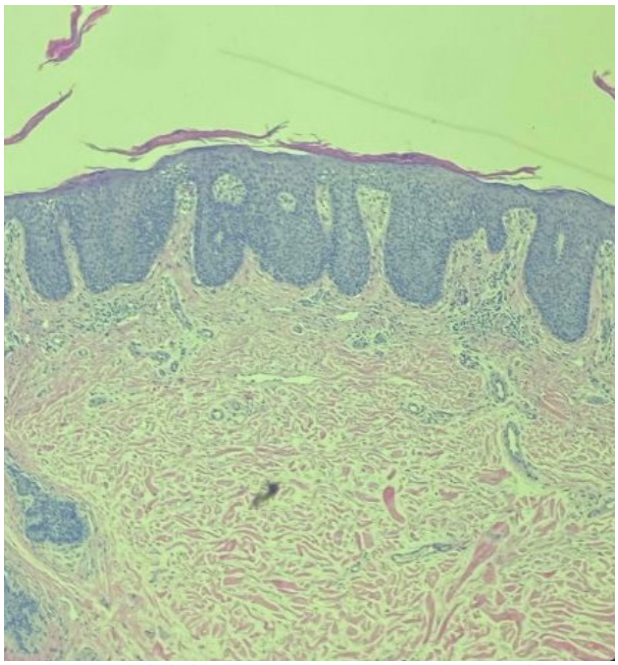

Figure 3. Epithelial alterations consistent with psoriasiform dermatitis including confluent parakeratosis, mounded neutrophils and regular acanthosis.

DISCUSSION

Erythrodermic psoriasis (EP) is a rare, potentially life-threatening, systemic manifestation of psoriasis. The pathophysiology of EP is poorly understood but is thought to involve Th1, Th2, and Th17-related pathways and their relationship with pro-inflammatory cytokines.[8] Clinical suspicion of EP should arise in a patient presenting with generalized inflammatory erythema

affecting between 75-90% of the total body surface area. Confirmatory diagnosis can be achieved via skin biopsy and histopathologic examination demonstrating epidermal perivascular infiltration of lymphocytes and eosinophils, hyperkeratosis, and dilated capillaries.[2] When confirmation by biopsy is desired, care should be taken to take the skin sample from the center of an active lesion, as the characteristic histopathology is most distinguishable from these sites and limits otherwise unnecessary repeat biopsy.[7] Patients with EP are at increased risk for bacteremia and sepsis caused by common skin pathogens such as Staphylococcus aureus. Left untreated, EP can result in a number of complications including peripheral edema, high-output heart failure, and acute kidney failure. The skin barrier dysfunction observed in EP can also result in insensible fluid losses and electrolyte imbalances.[9] Useful tests and laboratory markers for workup of EP may include EKG, comprehensive metabolic panel, complete blood count, C-reactive protein, erythrocyte sedimentation rate, and institution-specific septic-protocol measures such as lactate dehydrogenase.

Initial management of suspected EP should include correction of electrolyte and fluid abnormalities, empiric treatment of secondary infections, and monitoring for symptoms of hypothermia. For unstable or acutely ill patients with EP, the first-line therapy as published by the US National Psoriasis Foundation consists of either cyclosporine or infliximab, while stable patients are recommended either methotrexate or acitretin.[2,6]

The decision of one drug over the other is a shared process with the patient and should consider aspects of the patient's health. Cyclosporine is an immunosuppressive agent whose action is mediated via inhibition of calcineurin, with a rapid onset of action that can be used for unstable patients with suspected EP.[2] While cyclosporine provides rapid reversal of symptoms, relapse is common after discontinuation of the drug. Furthermore, there are numerous drug-drug interactions, including statins, thiazide, and potassium-sparing diuretics, warfarin, and NSAIDs. With long-term use, renal fibrosis and irreversible nephrotoxicity become a major concern.[10] Infliximab, a TNF-α inhibitor, has also been approved as monotherapy for patients diagnosed with EP. Like cyclosporine, infliximab has a rapid onset of action, and its use has led to a host of biologic treatments being explored for treatment of EP. However, this drug predisposes patients to significant infection risk and should not be initiated in patients with active infection, which may be an especially large concern in EP.[10] Patients should have their tuberculosis status assessed clinically prior to trialing this drug. Finally, infliximab is relatively contraindicated in patients with a history of lymphatic malignancy, NYHA class III or IV congestive heart failure, or untreated hepatitis B infection.

For stable patients, methotrexate and acitretin have proven efficacious. Methotrexate, a dihydrofolate reductase inhibitor, has a slower onset of action than those drugs recommended for unstable patients. It should be provided with a folic acid supplement. Bone

	Erythrodermic Psoriasis	Atopic Dermatitis
Mean Age	53.7 years	3-6 months
Male: Female Ratio	3:1	1:1
Prevalence	0.03 – 0.0675%	10.7% (Pediatric) 7.2% (Adult)
Pathophysiology	T_H1 and T_H17 mediated inflammatory response[12]	Type IV hypersensitivity response, T_H2 mediated[12]
Patient Presentation	Erythematous patches and plaques, with possible mild pruritus, burning, desquamation, and >75% skin involvement	Severely pruritic erythematous patches on the flexural surfaces
Diagnosis	Clinical, biopsy is confirmatory	Clinical
Histopathology	Elongation of rete ridges, elongation of dermal papillae, acanthosis, spongiosis, hyperkeratosis and parakeratosis (may be absent in areas of desquamation), perivascular infiltrate of eosinophils and neutrophils	Tissue spongiosis within the epidermis with overlying parakeratosis, lymphocytic infiltrate, with occasional histiocytes, neutrophils, and eosinophils[13]
Treatment	Unstable: Cyclosporine, infliximab Stable: Methotrexate, acitretin	First line: Topical corticosteroids Second line: Topical calcineurin inhibitors[14]

Table 1. Comparison of erythrodermic psoriasis and atopic dermatitis.

marrow suppression and hepatotoxicity are known possible complications of long-term methotrexate use. Patients should be monitored for signs of infection, anemia, and have their liver function assessed.[2,6,10] Acitretin is a non-immunosuppressing therapy and is a vitamin A derivative retinoid. By regulating keratinocyte proliferation and local inflammation, they appropriately address the root cause of EP. However, this drug is teratogenic and should be avoided in women of childbearing age who are not on any form of birth control. Acitretin is also contraindicated in patients with severe kidney or liver disease. Other notable side effects include brittle nails, cheilitis, xerosis, and possible hair loss.[2,6,10] The role of topical therapy has become less common due to the advent of more potent systemic agents.

This is a disease that warrants prompt recognition and treatment given its morbidity and risk of patient mortality. While EP may be at first mistaken for atopic dermatitis as in our patient, EP should remain on the differential, particularly when there is >75% estimated skin involvement. Atopic dermatitis affects the pediatric population more commonly, appearing as erythematous lesions commonly on the flexural surfaces and is known to be intensely pruritic.[11] While widespread skin involvement is possible, this is a less common presentation of atopic dermatitis. Intense pruritic can also help physicians distinguish between the two ailments. While EP can have some degree of pruritus, it is not known to be severe, and often allodynia or burning pain are associated with EP.[8] The skin itself may provide further clues to increasing clinical suspicion for EP over atopic dermatitis, as EP commonly has broad desquamation not seen in atopic dermatitis. The primary differences between these two similar but distinct pathologies are summarized in Table 1. All considered, in an elderly patient presenting with erythematous rash with >75% skin involvement, even when pruritus is present, EP should remain high on the differential, particularly in patients with a known history of psoriasis.

CONCLUSION

A high index of suspicion must be maintained when patients present with clinical symptoms consistent with erythrodermic psoriasis. A missed diagnosis can expose patients to treatments that are ineffective and hold the possibility of worsening their condition. Care must be taken when collecting tissue for biopsy in patients presenting with new psoriatic plaques. These biopsies should be taken centrally from active lesions. This will improve patient outcomes and decrease the risk of missing rare diagnoses, such as in our patient who was initially diagnosed with atopic dermatitis but later discovered to have erythrodermic psoriasis.

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CONFLICTS OF INTEREST

All authors declare no conflicts of interest.