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Erythrodermic Psoriasis and Staph-Infective Endocarditis—A Conundrum in Succession

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Abstract

Erythrodermic psoriasis is a rare subtype of psoriasis vulgaris that presents with diffuse erythema and desquamation over greater than 75% of the body's surface area. We present a case of a 57-year-old male who was admitted with a diffuse, erythematous scaly rash covering his entire body, with associated subjective fevers. Skin biopsy revealed erythrodermic psoriasis, and blood cultures were positive for methicillin-sensitive *Staphylococcus aureus*. Echocardiogram revealed a mitral valve vegetation. Clinical improvement was achieved with intravenous antibiotic administration and topical corticosteroids without the use of immunomodulators.

Keywords

erythrodermic psoriasis, methicillin-susceptible *Staphylococcus aureus* (MSSA), infective endocarditis, bacteremia

Introduction

Erythrodermic psoriasis is a rare, life-threatening form of psoriasis vulgaris characterized by diffuse erythema, pain, scaling, and flaking of the skin of which greater than 75% of the body's surface area is involved. Of the many types of psoriasis, erythrodermic psoriasis is the least common type, with a 1% to 2.25% prevalence.[1] A history of psoriasis vulgaris is the most common risk factor for developing erythrodermic psoriasis.[2] Furthermore, systemic glucocorticoids, abrupt discontinuation of systemic anti-psoriatic medications,[2,3] and infections[4] are known triggers of erythrodermic psoriasis. Due to excessive cutaneous vasodilation, high-output heart failure with multiple organ dysfunction could occur.[1] Rapid turnover of epidermal cells requiring greater metabolic demands in combination with the inability to thermoregulate leads to water loss and electrolyte derangements. Patients may present with edema due to protein loss via skin exfoliation. Furthermore, due to the loss of the skin barrier, these patients are more susceptible to infections from common skin flora, such as *Staphylococcus aureus.* Prompt diagnosis and treatment is crucial to avoid complications associated with defects in thermoregulation, and hemodynamic and metabolic instability.[1]

Case Presentation

A 57-year-old male presented to the emergency department with diffuse, scaly, flaky erythematous pruritic skin rashes which began abruptly over his entire body including his head and neck about a month ago. He had one similar episode in the past, which was self-limiting, and he never sought medical attention. Three days prior to presentation in this episode, he also noticed to have fevers and found a left-arm spontaneous sloughing open wound with purulent discharge.

On arrival, the patient's vital signs were significant for tachycardia of 140 beats per minute and a fever of 39.4°C. Physical examination revealed a diffuse erythematous rash with large sheets of exfoliation and scaling which covered almost his entire body (Figure 1). There was an open 8-cm wound on the anterior proximal of the left arm with purulent discharge (Figure 2). He had poor dentition with a normal fundoscopic examination. His cardiac examination revealed tachycardia without any murmurs, gallops, or rubs. Laboratory results showed lactic acid of 6.2 mmol/L, C-reactive protein (CRP) of 1.31 mg/dL, and erythrocyte sedimentation rate (ESR) of 40 mm/h. White blood count was within the normal range (Table 1). Urinalysis was negative for hematuria, proteinuria, and red blood cell casts. Skin biopsy was consistent with psoriasiform dermatitis, and in conjunction with the classic clinical presentation, the diagnosis of erythrodermic psoriasis was made. He was placed on

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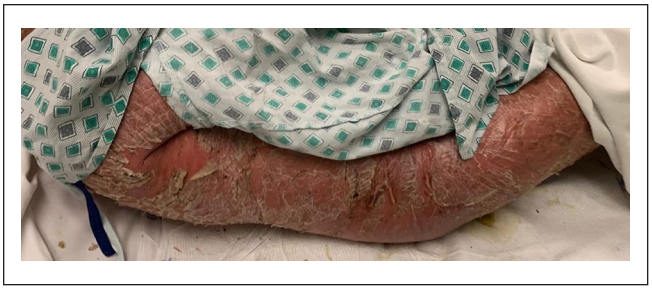

Figure 1. Diffuse erythematous rash with large sheets of exfoliation and scaling covering entire body.

topical triamcinolone 0.1%. Blood cultures were obtained, and he was started on vancomycin and piperacillin-tazobactam empirically. His depleted electrolytes were replaced. Cultures from blood and open wound both grew methicillin-sensitive *Staphylococcus aureus* (MSSA), and his antibiotics were de-escalated to nafcillin. Transesophageal echocardiogram revealed a 0.14 cm × 1.57 cm vegetation on the atrial side of the anterior mitral valve (Figure 3). Systemic treatment for his erythrodermic psoriasis was deferred due to the presence of bacteremia and infective endocarditis. Clinical improvement of the skin lesions began slowly after 6 days of topical treatment and proper skincare. His surveillance blood culture became sterile. He was discharged with home health to finish a total of 42 days of nafcillin. He was scheduled to be seen by a local dermatologist but lost to follow-up.

Figure 2. An oblique-shaped open wound (8 cm in length) on the anterior proximal of the left arm with purulent discharge. Erythematous rash with large sheets of exfoliation and scaling is also seen in upper arm and anterior chest wall.

Discussion

Erythrodermic psoriasis is a rare variant of psoriasis characterized by diffuse erythema and desquamation of the skin covering most of the body's surface and could cause severe morbidity and mortality when left untreated.

The exact immunopathogenesis of erythrodermic psoriasis is yet to be discovered. Insight is mainly based on small studies involving comparisons to psoriasis vulgaris. The classic silvery plaques seen in psoriasis are a result of keratinocyte hyperproliferation secondary to activated T cells infiltrating the skin.[5] Erythrodermic psoriasis involves a greater proportion of Th2 differentiation compared with Th1 with subsequent increased levels of IL-4, IL-10, and IgE.[6] Increased levels of TNF-alpha have also been observed in patients with this condition, and thus, TNF-alpha inhibitors have been efficacious in the treatment of erythrodermic psoriasis.[7] Further investigation of the pathogenesis and the dysregulation of inflammatory pathways and related cytokines can be useful in the discovery of more targeted therapies.

As discussed by Singh et al, the typical presentation of erythrodermic psoriasis includes erythematous, desquamative, and pruritic psoriatic plaques, like this case which was almost covering his entire body. Laboratory studies can be significant for electrolyte imbalances, anemia, high ESR and CRP levels, and leukocytosis. This patient had hypokalemia, hyponatremia, hypophosphatemia, hypomagnesemia, anemia, and elevated CRP and ESR (Table 1). Patients can present with additional symptoms of fever, tachycardia, dehydration, malaise, allodynia, and cachexia.[1] This case presented with fever and tachycardia perhaps due to known symptoms of erythrodermic psoriasis and his bacteremia and infective endocarditis combined.

Cutaneous vasodilation due to widespread inflammation and decreased venous return can lead to increased cardiac output, and potentially progress to high-output heart failure, followed by multiple organ dysfunction and shock.[1] Fortunately, this case did not show any evidence of heart failure with an ejection fraction of 60% and had no organ failure. Histologic examination of the skin lesions can confirm the diagnosis if physical examination raises high suspicion of erythrodermic psoriasis. Hyperkeratosis and epidermal perivascular lymphocytic and eosinophilic infiltration with dilated capillaries are diagnostic histological findings.[8]

Stabilization of electrolytes, protein, and fluid, as well as hypothermia prophylaxis, is the first step in the management of erythrodermic psoriasis.[2] Our patient had elevated lactic acid levels, which normalized after intravenous fluid resuscitation. Topical steroids and vitamin D analogues are less commonly used as more targeted and effective therapies are

Table 1. Laboratory Values on Admission With Reference Values.

Laboratory values		
		Reference range
Lactic acid	6.2 mmol/L	0.4-2.0 mg/dL
CRP	1.31 mg/dL	<0.30 mg/dL
ESR	40 mm/h	<20 mm/h
WBC	7.6×10^3/μL	$4.5\text{-}11.0 \times 10^3$/μL
Hemoglobin	10.2 g/dL	13.2-17.4 g/dL
AST	54 unit/L	15-37 unit/L
Albumin	2.6 g/dL	3.4-5.0 g/dL
Sodium	134 mmol/L	136-145 mmol/L
Potassium	3.2 mmol/L	3.5-5.1 mmol/L
Calcium	7.6 mg/dL	8.5-10.1 mg/dL
Phosphorus	2.4 mg/dL	2.5-4.9 mg/dL
Magnesium	1.3 mg/dL	1.8-2.4 mg/dL

Abbreviations: CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; WBC, white blood cell; AST, aspartate aminotransferase.

available.[8] Systemic agents, such as second-generation retinoids, methotrexate, and cyclosporine, and biologics including TNF-alpha inhibitors, and IL-12, IL-23, and IL-17 inhibitors,[1] have shown greater clinical improvement.[8]

The prevalence of staphylococcal colonization in patients with psoriasis is 35.3%[9] which is higher than in healthy patients. As a result, secondary infections of the skin and subsequent bacteremia with *S aureus* and Group A Streptococcus are not uncommon in psoriatic patients and require attention and appropriate antibiotics.[3] One study identified that 6 out of 7 patients with guttate psoriasis had ribosomal DNA of streptococcus, and staphylococcus was detected in 9 out of 13 patients with psoriasis.[10]

This relationship has not been studied in erythrodermic psoriasis, but diffuse exfoliation and scaling may result in an even higher chance of skin and subsequent penetration to the bloodstream compared with other forms of psoriasis as seen in this report. Our hypothesis is that he had a high chance of colonization with MSSA and breaching in skin barrier at the same time, which triggered the penetration to his bloodstream. Furthermore, the repetitive or larger inoculum and duration of bacteremia perhaps contributed to the formation of vegetation on his mitral valve. This chain of events was presumably the sequelae of his untreated erythrodermic psoriasis.

On the contrary, the risk of infection can also increase if the patient is being treated with immunosuppressive medications.

Immunomodulators are the first-line treatment for erythrodermic psoriasis with known increased chance of infections even further than was discussed.[1] We had to forgo such treatment for more conservative and supportive measures secondary to the patient's confounding bacteremia and infective endocarditis. Although our choice of treatment with topical corticosteroids and wet dressings was more conservative than immunomodulators, we believe it achieved reasonable comparable results in the patient's resolution of symptoms and reversal of the disease process. The patient's pruritis, erythema, and skin flaking significantly improved after 6 days of topical treatment alone.

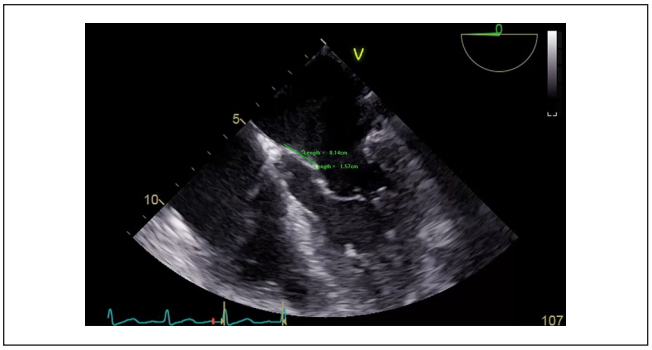

Figure 3. Transesophageal echocardiogram revealed a 0.14 cm × 1.57 cm vegetation on the atrial side of the anterior mitral valve.

Conclusion

Erythrodermic psoriasis can lead to life-threatening complications such as infective endocarditis presumably from a skin source. In cases of serious systemic infections when prolonged antibiotics are needed, the treatment options become limited, challenging, and delayed.

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Ethics Approval

Ethical approval to report this case was obtained from the Kern Medical Institutional Review Board (Approval ID: 20059).

Informed Consent

Written informed consent was obtained from the patient for their anonymized information and photographs to be published in this article.

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References

1. Singh RK, Lee KM, Ucmak D, et al. Erythrodermic psoriasis: pathophysiology and current treatment perspectives. *Psoriasis (Auckland)*. 2016;6:93-104. doi:10.2147/PTT.S101232.
2. Boyd AS, Menter A. Erythrodermic psoriasis. *J Am Acad Dermatol*. 1989;21:985-991.
3. Rosenbach M, Hsu S, Korman NJ, et al. Treatment of erythrodermic psoriasis: from the medical board of the National Psoriasis Foundation. *J Am Acad Dermatol*. 2010;62:655-662. doi:10.1016/j.jaad.2009.05.048.
4. Morar N, Willis-Owen SA, Maurer T, et al. HIV-associated psoriasis: pathogenesis, clinical features, and management. *Lancet Infect Dis*. 2010;10:470-478. doi:10.1016/S1473-3099(10)70101-8.
5. Nair PA, Badri T. *Psoriasis*. Treasure Island, FL: StatPearls; 2021.
6. Li LF, Sujan SA, Yang H, Wang W-H. Serum immunoglobulins in psoriatic erythroderma. *Clin Exp Dermatol*. 2005;30:125-127. doi:10.1111/j.1365-2230.2004.01717.x.
7. Lee WK, Kim GW, Cho HH, et al. Erythrodermic psoriasis treated with golimumab: a case report. *Ann Dermatol*. 2015;27:446-449. doi:10.5021/ad.2015.27.4.446.
8. Prystowsky JH, Cohen PR. Pustular and erythrodermic psoriasis. *Dermatol Clin*. 1995;13:757-770.
9. Ng C, Huang Y, Chu C, Wu TC, Liu SH. Risks for *Staphylococcus aureus* colonization in patients with psoriasis: a systematic review and meta-analysis. *Br J Dermatol*. 2017;177:967-977. doi:10.1111/bjd.15366.
10. Munz OH, Sela S, Baker BS, Griffiths CEM, Powles AV, Fry L. Evidence for the presence of bacteria in the blood of psoriasis patients. *Arch Dermatol Res*. 2010;302:495-498. doi:10.1007/s00403-010-1065-0.