Case Report

Erythrodermic Psoriasis Managed with Risankizumab

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Keywords
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Abstract
Erythrodermic psoriasis (EP) is a severe, often refractory, variant of psoriasis. Due to the high morbidity and mortality rate associated with EP and other causes of erythroderma, they are often classified as dermatologic emergencies. EP is usually a therapeutic challenge, where topical and conventional systemic therapies have yielded a less than satisfactory result in several patients. Furthermore, there are a limited number of studies evaluating other therapeutic modalities, such as biologic agents, with no clear treatment guidelines. In this case report, we present a patient who was diagnosed as a case of EP and showed an impressive response to risankizumab.

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Introduction

Erythrodermic psoriasis (EP) is a rare, life-threatening subtype of psoriasis that affects 75–90\% of the body surface area (BSA) \cite{1}. Due to impairment of skin barrier function, EP can be associated with complications such as secondary infections and sepsis (specifically with \textit{Staphylococcus aureus}) \cite{1–3} and loss of transepidermal fluid resulting in dehydration, acute kidney injury, and shock \cite{1, 2}. Other complications include electrolyte abnormalities,
acute respiratory distress syndrome, thermal dysregulation and hypothermia, severe anemia, and high-output congestive heart failure [1, 2]. The aforementioned reasons give grounds for the need for EP to be managed rapidly and systemically. Traditionally, conventional systemic agents such as cyclosporine, acitretin, and methotrexate were used as first-line therapies for EP, alone or in combination [4]. With the introduction of revolutionary biologic therapies, improved treatment rates and higher safety profiles are seen in patients with psoriasis, making biologic agents the go-to medications for dermatologists in many resistant cases. However, there are a limited number of studies evaluating the efficacy of these agents in managing EP. A recent systematic review evaluating these biological agents states that there is limited high-quality evidence showing one agent superior to another. They recommended the use of infliximab and ustekinumab as first-line agents in acute, severe cases of EP [5]. Both reviews found in the literature contain studies that were conducted before the Food and Drug Administration (FDA) approval of risankizumab. Risankizumab is a fully human IgG monoclonal antibody that inhibits the p19 subunit of IL-23 [6]. In 2019, risankizumab received FDA approval for the treatment of moderate to severe psoriasis [7]. We present a patient with EP who showed complete resolution with risankizumab despite a suboptimal response to other systemic agents.

**Case Report/Case Presentation**

A 48-year-old Saudi man presented to our clinic in March 2021 with widespread erythematous and desquamated plaques. The patient had already been diagnosed with chronic plaque psoriasis since 2014, with no joint involvement. Before his diagnosis, the patient was medically free, had no medications or herbal supplements, and, as far as he could recall, had not developed infections. He has been a smoker for more than 20 years. His sister has similar scaly lesions on her hands but was never officially diagnosed with psoriasis. His lesions first appeared on his hands and feet, for which he received topical calcipotriol/betamethasone and emollients to be applied daily. Despite applying the topical treatment compliantly, his skin lesions continued to progress and began to affect his knees, elbows, wrists, and scalp. The patient had a chronic remitting course and was never offered phototherapy or systemic treatment. Before coming to our clinic, he experienced a flare of his condition with new lesions appearing all over his body. This flare began a few days after the patient started an antibiotic course (he could not recall the antibiotic name) for a urinary tract infection. The lesions progressed within 2 weeks and were associated with minimal pruritus. He also complained of subjective fever, myalgias, and arthralgias, which were sometimes severe enough to prevent him from sleeping. He then received a single dose of secukinumab, 1 week prior to presenting to our clinic.

On physical examination, there were generalized erythematous scaly plaques affecting his scalp, face, trunk, abdomen, bilateral lower, and upper extremities, affecting 90% of his BSA (shown in Fig. 1–3). Nail pitting was observed, but no other psoriatic nail changes were found. There was no swelling or tenderness in the joints. A punch biopsy taken from a lesion on his right arm revealed an intracorneal neutrophilic collection with parakeratosis, irregular psoriasiform hyperplasia with spongiosis of the epidermis, focally decreased granular layer, perivascular lymphocytic infiltrate with some eosinophils in the superficial dermis. Then it was diagnosed as a case of EP.

We started the patient on risankizumab (150 mg on day 0, week 4, and then every 12 weeks). He was not continued on secukinumab as it was unavailable in our institute. During treatment, the patient reported an initial worsening of muscle and joint pain. However, these symptoms quickly subsided with subsequent doses. By the third dose, the patient showed a
Fig. 1. The pictures on the left show diffuse erythema with overlying silvery scaly plaques over the anterior trunk and upper extremities, occupying >90% of the body surface area, prior to treatment with risankizumab. The pictures on the right show some post-inflammatory hyperpigmentation with no active skin lesions, taken post-treatment with risankizumab.

Fig. 2. The pictures on the left show diffuse erythema with overlying silvery scaly plaques over the posterior trunk and upper extremities, occupying >90% of the body surface area, prior to treatment with risankizumab. The pictures on the right show some post-inflammatory hyperpigmentation with no active skin lesions, taken post-treatment with risankizumab.
complete resolution of his skin lesions (shown in Fig. 1–3) and reported a resolution of pruritus, myalgias, and arthralgias.

**Discussion/Conclusion**

EP is a rare variant of psoriasis, affecting 1–2.25% of psoriasis patients [8]. Psoriasis is actually the most common cause of erythroderma, accounting for 25% of all cases of erythroderma [8, 9]. Cutaneous findings of EP typically present as generalized (involving at least 75% of the BSA) erythema, edema, and pruritis with possible palmoplantar involvement and psoriatic nail changes [8]. Patients may also present with systemic symptoms such as fever, chills, tachycardia, malaise, arthralgias, myalgias, a change in bowel habits, and weight changes. Laboratory derangements such as anemia, elevated C-reactive protein, and erythrocyte sedimentation rate, and electrolyte abnormalities could be found as well [8]. EP patients are also more prone to develop infectious complications of the skin and blood, with *S. aureus* and Group A streptococcus, which can lead to more serious infections such as cellulitis, erysipelas, pneumonia, endocarditis, osteomyelitis, and sepsis [8, 9]. The pathogenesis of EP is not completely understood. A genetic basis for EP has not been clarified, unlike other variants of psoriasis [8]. Immunologically, an imbalance in Th1/Th2 levels has been demonstrated [10]. Higher levels of the transcription factor GATA-3, which results in Th2 differentiation, and higher levels of IL-4, a Th2 cytokine, were found in patients with EP, which suggests increased Th2 levels [10]. This justifies the high levels of IgE found in patients with EP [10]. Th17 cells were also found to be the most prominent cells involved in EP after Th2 cells [11]. Environmental triggers were also implicated in the pathogenesis of EP, including but not limited to emotional stress, trauma, systemic illnesses such as leukemia or infections with certain viruses like human immunodeficiency virus, exposure to certain drugs or chemicals, and sudden withdrawal of certain medications such as systemic steroids (rebound phenomenon) [8].
Treatment guidelines for EP are lacking. Consensus guidelines were published in 2010 by the National Psoriasis Foundation Medical Board of the USA advocate the use of conventional therapies such as acitretin, methotrexate, cyclosporine, and infliximab [4]. Cyclosporine and infliximab were preferred in acute and unstable cases [4]. In resistant cases, etanercept or combination therapy was recommended [4]. Recent studies suggest that biologic agents may be a better option for EP. A systematic review completed a comprehensive analysis of 43 studies in the treatment of EP with biological agents. They concluded that the use of infliximab or ustekinumab is recommended as first-line agents in severe, acute cases of EP. However, both reviews found in the literature included studies conducted before the FDA approval of risankizumab [2, 5]. Risankizumab is a humanized IgG monoclonal antibody that selectively inhibits the p19 subunit of IL-23 [6]. IL-23 triggers the differentiation of Th17 and Th22 cells [6]. As mentioned previously, Th17 cells were found to be involved in the pathogenesis of EP, thereby justifying the use of risankizumab in EP patients. Risankizumab was also FDA approved for the management of moderate-to-severe psoriasis in April 2019 [7]. And in March of 2019, risankizumab achieved global approval in Japan for management of psoriasis vulgaris, psoriatic arthritis, generalized pustular psoriasis, and EP in adults [12]. Due to the complications associated with EP, rapid and effective management is indicated, making biologic agents, such as risankizumab a suitable choice.

Statement of Ethics

The research was conducted ethically in accordance with the Declaration of Helsinki. Ethical approval was not required for this study in accordance with local/national guidelines since this is a single case report without identifying information about the patient. Written informed consent was obtained from the patient for publication of the details of the case and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Abdulmajeed Alajlan investigated and managed the case, provided critical feedback, and approved the final version of this manuscript. Abdulaziz Madani investigated and managed the case as well, provided critical feedback, and approved the final version of this manuscript. Tala Ammar Qadoumi worked with both Abdulmajeed Alajlan and Abdulaziz Madani in investigating and managing the case, wrote the pathologic description and manuscript, and provided critical feedback. Alhanouf Aljaloud communicated with the patient and wrote the pathologic description and case presentation. Mohammed Alessa obtained the written consent from the patient for this report and helped write the pathologic description and case presentation.
Data Availability Statement

The patient data are not publicly available on legal or ethical grounds. All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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