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CASE REPORT

Successful Treatment of Severe and Refractory Eosinophilic Pustular Folliculitis with Dupilumab: A Case Report with Two-Year Relapse-Free Follow-Up

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Background: Eosinophilic pustular folliculitis (EPF) is a rare chronic inflammatory skin disease characterized by eosinophilic infiltration and recurrent sterile pustules. Current treatments are often ineffective, and the disease frequently relapses.

Methods: We report a case of a 71-year-old female with severe EPF and a 17-year history of recurrent pruritic papulopustular eruptions. Dupilumab was administered subcutaneously with a 600 mg loading dose followed by 300 mg every two weeks over two treatment courses, separated by a five-month loss to follow-up.

Results: After dupilumab therapy, the patient's skin lesions completely resolved, accompanied by a marked decrease in peripheral eosinophil counts. During a 2-year follow-up, no recurrence was observed.

Conclusion: Dupilumab demonstrated significant efficacy and safety in this severe and refractory EPF case, resulting in long-term remission without relapse, and highlighting its potential as a novel treatment option for EPF.

Keywords: eosinophilic pustular folliculitis, eosinophilic diseases, therapy, dupilumab, biologic agent

Background

Eosinophilic pustular folliculitis (EPF) is a rare, non-infectious, eosinophil-dominated inflammatory skin disorder, first described by Ofuji in 1970.[1] It is characterized by recurrent follicular papules and pustules, typically affecting seborrheic areas such as the face, trunk, and limbs.[2] The condition may also involve the palmoplantar regions and the nails occasionally.[3,4] Histologically, EPF is marked by eosinophil infiltration around pilosebaceous units, accompanied by eosinophilic microabscesses.[5,6] The etiology remains unclear, but potential triggers include hypersensitivity reactions, fungal infections, immune dysfunction, and pregnancy.[7] EPF has three clinical variants: classic EPF, immunosuppression-associated (IS-EPF, often linked to HIV infection or hematologic malignancies), and infancy-associated (I-EPF).[8,9] The condition is more frequently reported in Asian populations, with classic EPF showing a predilection for middle-aged men, while infantile cases are less common and typically self-limited.[10] All variants show eosinophil-rich skin inflammation with peripheral eosinophilia.[11] It is important to distinguish EPF from papuloerythroderma Ofuji, a separate entity also first reported by Ofuji, which presents with generalized erythroderma and the characteristic "deck-chair sign".[1,12] Despite its rarity, the relapsing nature and limited response to conventional therapies highlight the need for alternative treatment strategies.

Apart from classic EPF, which responds well to systemic indomethacin, treatment for all three types of EPF primarily relies on topical and systemic corticosteroids. In addition, topical tacrolimus can be used for non–HIV-associated IS-EPF.[13] However, some cases of EPF are severe, refractory, and prone to relapse, highlighting the need for new therapeutic approaches.[14] Advances in biologic therapies have broadened the therapeutic options available for EPF. Currently, biologics reported for EPF include dupilumab, anti-IL-5 antibodies, and JAK inhibitors.[15–17] Dupilumab, an IL-4 receptor α antagonist,

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inhibits IL-4 and IL-13 signaling pathways central to eosinophil-driven inflammation. Clinical evidence for the use of anti-IL -5 antibodies, such as mepolizumab, in EPF remains limited. Compared with the broad downstream inhibition of JAK inhibitors, dupilumab offers a more precise targeted mechanism and a more favorable safety profile, making it more suitable for long-term management of chronic disease, particularly in patients who have failed conventional therapies. In recent years, scattered case reports have documented favorable outcomes in patients with refractory EPF, but reports on the use of dupilumab for its treatment remain limited.[5] We report a case of an elderly female with EPF who showed significant clinical improvement after dupilumab therapy, with no recurrence noted during long-term follow-up.

Case Report

A 71-year-old female was admitted due to recurrent pruritic papulopustular eruptions on the inner limbs and abdomen for 17 years. Initially, the lesions presented as erythematous papules, followed by recurrent pustules that ruptured and formed black crusts, accompanied by severe itching and pain at the eroded sites. The symptoms recurred intermittently. The patient initially had mild symptoms and experienced relief after multiple treatments at other hospitals, including traditional Chinese medicine and topical therapies. Over the years, her symptoms gradually worsened. Six months prior, pustular lesions reappeared on her feet, and laboratory tests revealed eosinophilia and a positive antinuclear antibody (ANA) test. Before admission, the patient had been continuously taking methylprednisolone, which was prescribed at another hospital for a prior diagnosis of eosinophilic dermatosis. Two months before hospitalization, methylprednisolone was initiated at 20mg once daily and gradually tapered to 8mg once daily. Afterward, the patient developed generalized papules, pustules, and erosions in the intertriginous areas.

Upon admission, the patient presented with chronic, recurrent, pruritic follicular papules and pustules, predominantly involving the axillae, groin, vulva, and trunk (Figure 1a) laboratory tests revealed an eosinophil percentage of 30.2% and an absolute eosinophil count of $3.14\times10^{\wedge}9$/L. Considering possible diagnoses such as bacterial folliculitis, suppurative hidradenitis, papular necrotizing tuberculid, lymphomatoid papulosis, and other autoimmune disorders, we performed targeted evaluations. The ANA profile showed a titer of 1:1000 with a centromere pattern, positive SSA (Ro-52) and mitochondrial M2 antibodies, while IgE levels were within normal limits. Tests for vasculitis, autoimmune hepatitis, antiphospholipid syndrome autoantibodies, immunoglobulins, and complement levels were unremarkable, helping to exclude systemic autoimmune vasculitis or autoimmune hepatitis. HIV and syphilis testing were negative, ruling out these infectious causes. Chest CT revealed multiple fibrotic and interstitial changes in both lungs. A bone marrow biopsy demonstrated active hyperplasia with 15% eosinophils. Immunophenotyping, BCR-ABL testing, NGS, and chromosomal analysis revealed no malignancy, excluding lymphoma and myeloproliferative disorders. Skin lesion cultures grew only Escherichia coli, with no fungi or other bacteria present, and stool tests for parasites were negative, ruling out bacterial folliculitis, suppurative hidradenitis, and parasitic infection.

Skin biopsy revealed focal necrosis in the epidermis and dermis, with massive eosinophilic and partial neutrophilic infiltration (Figure 1b). Additional findings included epidermal acanthosis, spongiosis, and significant dermal edema. A dense eosinophilic infiltrate extended from the epidermis and dermis to the subcutaneous fat layer, with numerous eosinophils surrounding blood vessels, sweat glands, and hair follicles. Based on clinical and pathological findings, EPF was considered.

Treatment consisted of intravenous methylprednisolone (40 mg once daily), topical therapies, and doxycycline for its anti-inflammatory properties. A culture of biopsy tissue and exudate revealed the presence of Escherichia coli, and ceftazidime was initiated for infection management. After one week, the therapeutic response remained suboptimal (Figure 2a). With informed consent, dupilumab was initiated at a loading dose of 600 mg subcutaneously, followed by 300 mg every two weeks as maintenance therapy. One day after initiating treatment, the patient's skin lesions showed improvement, characterized by surface crusting. After four weeks, marked improvement in cutaneous lesions was observed, with a notable reduction in new pustule formation, with normalization of peripheral blood eosinophil counts (Figure 2a and b). At this point, as the patient experienced significant subjective improvement, she discontinued follow-up treatment for five months. Six months after discharge, she presented again with newly developed skin lesions (Figure 2a). Laboratory tests revealed an eosinophil count of $0.78\times10^{\wedge}9$/L, with no significant elevation in IgE levels. Therefore, dupilumab therapy was resumed, consisting of an initial 600 mg dose followed by 300 mg every two weeks for a total of fifteen weeks. Treatment was discontinued when the patient's skin lesions had nearly resolved, no new

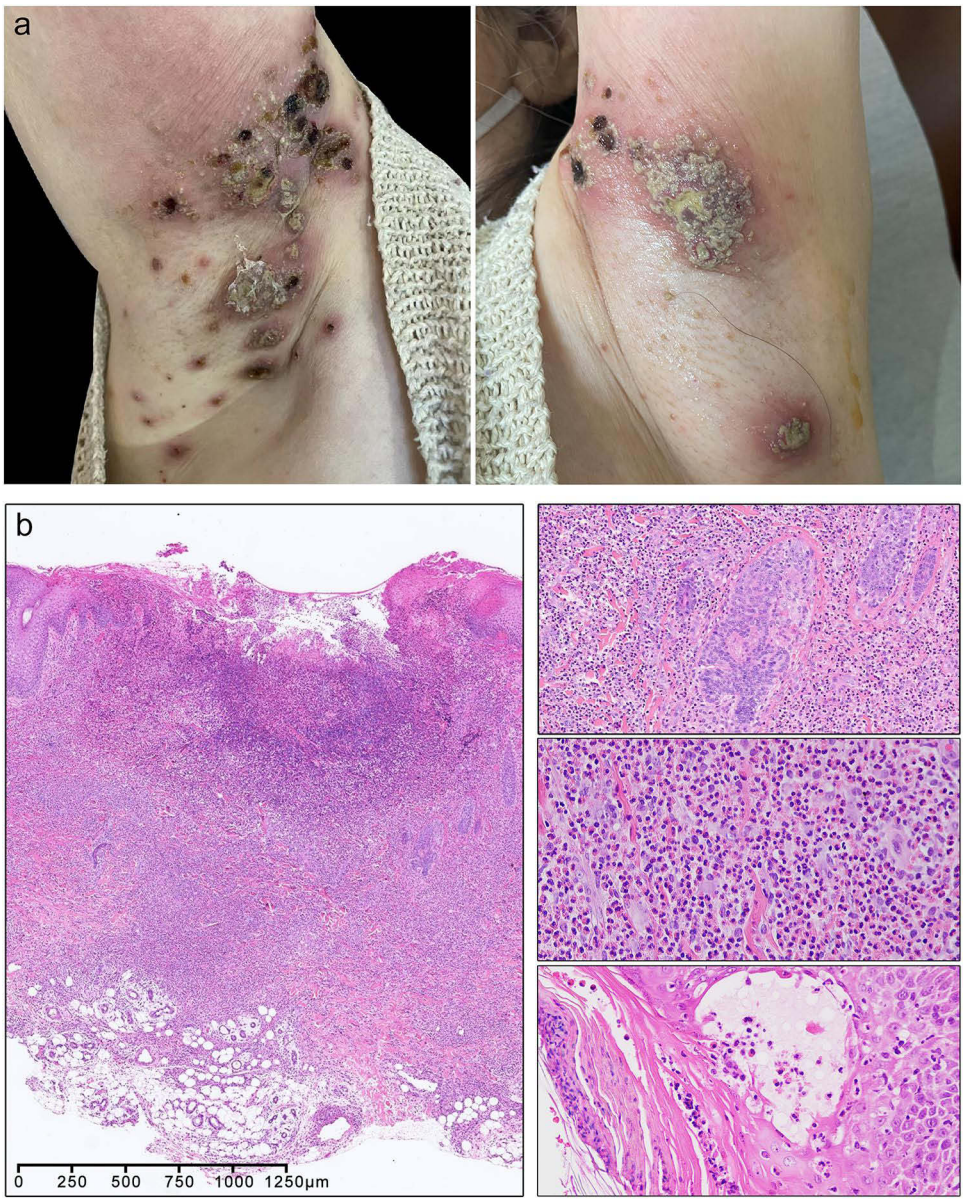

Figure 1 Clinical photographs and histopathological images of the patient. (**a**) Clinical photographs of the patient before and after dupilumab treatment, showing improvement in skin lesions following therapy. (**b**) Histopathological image of the lesions showed eosinophilic infiltration extending from the epidermis to the subcutaneous tissue and appendages, along with associated neutrophilic infiltration and spongiotic edema.

lesions had developed, and peripheral eosinophil counts had normalized. As the patient resided in a remote area, she was subsequently monitored through telephone follow-up, during which no evident recurrence of skin lesions was documented. During the two-year follow-up, no relapse occurred, and no significant adverse effects were observed during therapy (Figure 2c).

Discussion

EPF is a chronic inflammatory dermatosis characterized by sterile papulopustular eruptions centered on hair follicles, predominantly affecting the head, trunk, and extremities. Most patients exhibit peripheral eosinophilia, and some also show elevated serum IgE, indicating a role for type 2 inflammation. In this case, the patient presented with recurrent pruritic pustular lesions and marked eosinophilia, although IgE levels remained normal.

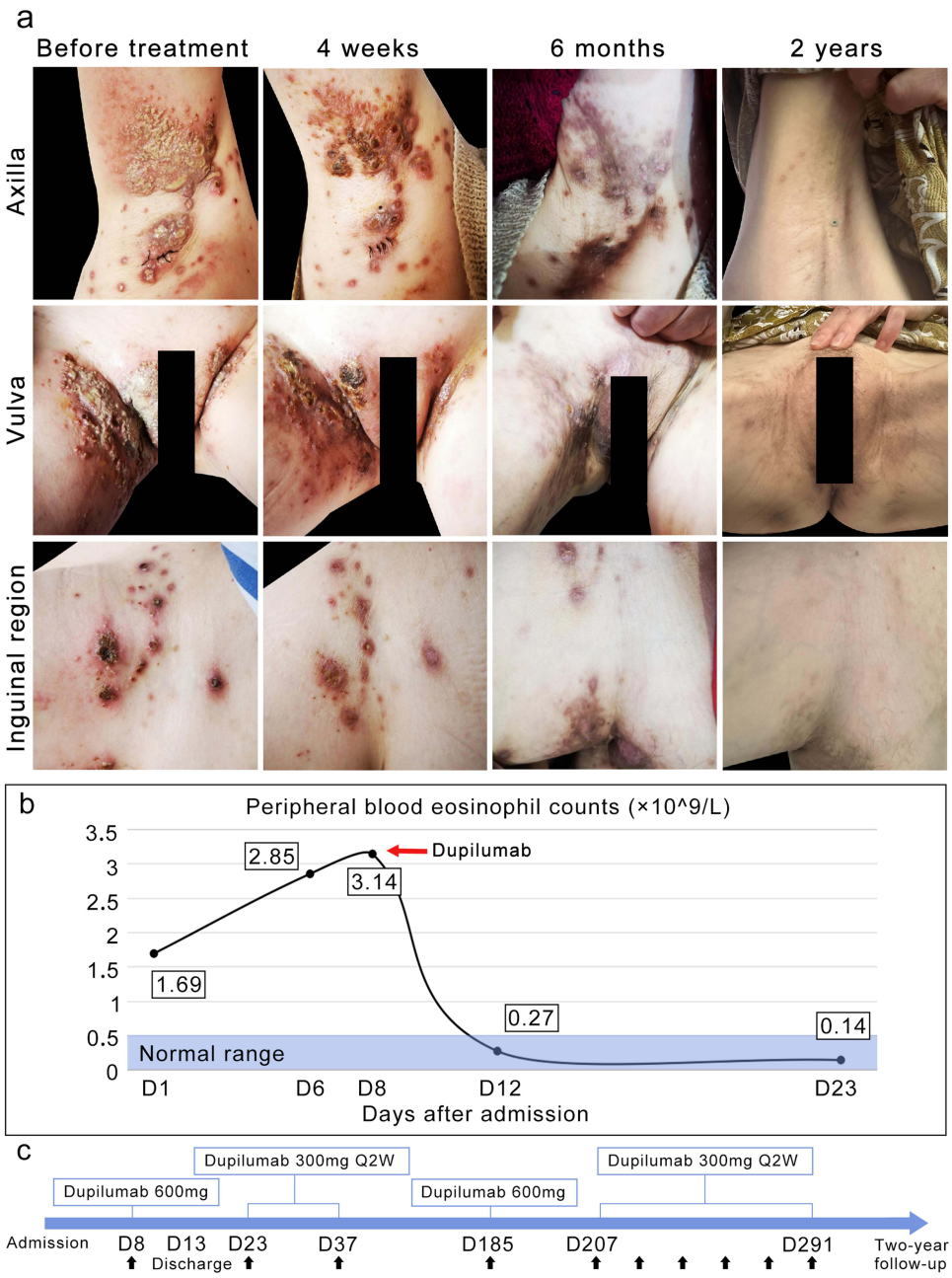

Figure 2 Changes in clinical symptoms and timeline of peripheral blood eosinophil counts and dupilumab treatment. (**a**) Clinical photographs of the patient's axillae, vulva, and groin taken before dupilumab treatment, and at 4 weeks, 5 months, and 2 years after dupilumab treatment. (**b**) Timeline of peripheral blood eosinophil counts during treatment. The red arrow indicates the initiation of dupilumab therapy. (**c**) Timeline of dupilumab treatment. The black arrows indicate the injections of dupilumab.

Based on these clinical and pathological findings, several differential diagnoses were considered, including bacterial folliculitis, suppurative hidradenitis, lymphomatoid papulosis, papular necrotizing tuberculid, parasitic infection, and hypereosinophilic syndrome (HES).[18–23] These were excluded through negative cultures, unremarkable bone marrow and molecular studies. The diagnosis was ultimately confirmed based on the patient's skin biopsy, which showed eosinophilic infiltration around hair follicles, a hallmark feature of EPF.[9,24]

Conventional treatments, such as indomethacin, systemic or topical corticosteroids, immunosuppressants like cyclosporine, and narrowband ultraviolet B (NBUVB) phototherapy, often provide only temporary relief and are associated with frequent relapses, especially when the dose is tapered.[13,24] The complex pathogenesis of EPF, involving eosinophilic infiltration, chemokines, and Th2-associated immune responses, suggests that therapies directed at a single pathway or

aimed solely at reducing eosinophil counts may be insufficient to achieve sustained disease control.[25] This patient had previously received systemic corticosteroids without satisfactory control, underscoring the limitations of traditional approaches.

Eosinophilia is primarily driven by type 2 helper T cells (Th2) and group 2 innate lymphoid cells (ILC2s) through the production of interleukin-5 (IL-5). In addition, interleukin-4 (IL-4) and interleukin-13 (IL-13) are key cytokines involved in the downstream signaling pathways of Th2-mediated inflammation. Dupilumab, a monoclonal antibody that blocks IL-4 and IL-13 signaling, has approved for atopic dermatitis and asthma, and has additionally been used to treat eosinophil-associated diseases including hypereosinophilic dermatitis, bullous pemphigoid, and pemphigus.[26,27] Currently, case reports on the use of dupilumab for the treatment of EPF remain limited, but a few studies have demonstrated its potential efficacy in refractory EPF.[5,28]

In our case, the patient initial treatment course consisted of a 600 mg loading dose followed by a single 300 mg dose administered two weeks later, totaling two injections. She was subsequently lost to follow-up. Five months later, the patient returned for follow-up and initiated a second course of dupilumab, beginning with a 600 mg loading dose, followed by 300 mg every two weeks, for a total of seven injections. Dupilumab induced rapid and sustained improvement, with resolution of lesions and normalization of eosinophil counts. The patient remained relapse-free during a two-year follow-up, highlighting dupilumab's potential for long-term disease control, resulting in a marked enhancement in quality of life and high satisfaction with treatment outcomes.

This case provides more robust clinical evidence supporting the potential efficacy of dupilumab in treating EPF, indicating that it may serve as an adjunctive therapy for Th2-mediated diseases. It should be noted that this report describes a single patient, and therefore the findings may not be generalizable to all individuals with EPF. While the observed clinical improvement with dupilumab is encouraging, further large-scale, randomized controlled trials are needed to confirm efficacy, assess long-term safety, and determine optimal dosing strategies.

Conclusion

Dupilumab therapy led to significant and sustained clinical improvement in a patient with severe eosinophilic pustular folliculitis who had shown poor response to conventional treatments. This case highlights the strong therapeutic potential of dupilumab in managing Th2-driven dermatologic conditions and supports its clinical application as a practical option for refractory EPF.

Data Sharing Statement

The data and material underlying this article are all available in the article.

Ethics Statement and Informed Consent

The study involving a human participant was reviewed and approved by the Sir Run-Run Shaw Hospital Ethics Committee. The patient provided written informed consent to participate in the study. Additionally, the patient provided written informed consent for the publication of all clinical details, images, and data included in this article. Institutional approval for publication of the case details was obtained from the Ethics Committee of Sir Run Run Shaw Hospital, Zhejiang University School of Medicine, Hangzhou, Zhejiang, People's Republic of China.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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