

Upadacitinib Induces Rapid Remission in Refractory Eosinophilic Pustular Folliculitis with Facial Involvement: A Case Report

Yanan WANG[†], Jinxuan SU[†]  and Hongzhong JIN^{*}

Department of Dermatology, State Key Laboratory of Complex Severe and Rare Diseases, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing 100730, China. *Email: jinhongzhong@263.net

[†]These authors contributed equally to this work.

Submitted May 16, 2026. Accepted after revision Aug 4, 2026

Published Sept 2, 2026. DOI: 10.2340/actadv.v106.adv-2026-0701 Acta Derm Venereol 2026; 106: adv-2026-0701.

Eosinophilic pustular folliculitis (EPF), also referred to as Ofuji disease, is a rare inflammatory dermatosis characterized by recurrent sterile follicular papules and pustules with prominent eosinophilic infiltration (1). Although EPF typically involves the trunk and extremities, facial involvement is uncommon and may lead to frequent misdiagnosis as eczema, rosacea, seborrheic dermatitis or fungal infection (2). The pathogenesis of EPF remains incompletely understood but is thought to involve eosinophil-dominant immune dysregulation and type two inflammatory signalling. Conventional treatments, including corticosteroids, antihistamines, and immunosuppressants, often show variable efficacy and frequent relapse (3). Janus kinase (JAK) inhibitors have emerged as potential targeted therapies. While pan-JAK inhibitors have shown efficacy in isolated reports, evidence regarding selective JAK1 inhibition in EPF remains scarce. Herein, we report a case of EPF with facial involvement successfully treated with upadacitinib, a JAK1 inhibitor.

CASE REPORT

A 37-year-old Chinese man presented with a more-than-10-year history of recurrent pruritic eruptions on the trunk and extremities and a 2-month history of a progressively enlarging erythematous lesion on the left cheek. The facial lesion gradually expanded in an annular pattern, with a raised infiltrative border, scattered pustules along the erythematous margin, mild scaling and central atrophy with hyperpigmentation. The lesion was intermittently pruritic and significantly affected the patient's appearance. At another hospital, fungal examination revealed the presence of *Malassezia* spores without significant hyphal elements, and dermoscopy showed a dark erythematous background with focal white scales and follicular pustules. The patient was misdiagnosed with rosacea and seborrheic dermatitis and received oral hydroxychloroquine for 10 days, with no improvement.

Physical examination revealed a well-demarcated annular erythematous plaque on the left cheek near the periorbital region, with a slightly elevated border, scattered pustules along the erythematous margin, minimal scaling and central atrophy with hyperpigmentation (Fig. 1 A). Scattered erythematous

papules and nodules with excoriations were also present on the trunk and extremities (Fig. 1 D–G). Laboratory findings revealed mild leukocytosis $10.65 \times 10^9/L$ (reference range, $3.5\text{--}9.5 \times 10^9/L$) and peripheral eosinophilia ($0.63 \times 10^9/L$) (reference range, $0.02\text{--}0.5 \times 10^9/L$), while erythrocyte sedimentation rate, C-reactive protein, immunoglobulins, complement levels and autoantibody profiles were within normal limits. Screening for infectious diseases was negative.

Histopathological examination of the facial skin lesion demonstrated hyperkeratosis with focal parakeratosis and mild acanthosis. An inflammatory infiltrate composed of lymphocytes, histiocytes, neutrophils, and eosinophils was observed around superficial and deep dermal vessels and folliculosebaceous units. Eosinophilic infiltration into the follicular epithelium was observed, supporting the diagnosis of EPF (Fig. 1 H–J).

Initial treatment with oral glycyrrhizin compound, antihistamines, and topical tacrolimus and corticosteroids for 2 weeks resulted in partial improvement of truncal lesions; however, the facial lesion became darker and continued to expand. Moreover, the patient declined systemic corticosteroid therapy. Therefore, upadacitinib was started at a dose of 15 mg once daily. Within 2 weeks, the patient experienced marked relief of pruritus and flattening of the lesion (Fig. 1 B). Significant resolution of erythema and pustules was observed after 4 weeks. At 6-month follow-up, the lesion had almost completely resolved, leaving only post-inflammatory hyperpigmentation, with no evidence of recurrence during subsequent follow-up (Fig. 1 C).

DISCUSSION

Eosinophilic pustular folliculitis (EPF), also referred to as Ofuji disease, is a chronic relapsing inflammatory disorder characterized by sterile follicular papules and pustules with prominent eosinophilic infiltration (1). Although the disease most commonly affects seborrheic regions such as the trunk and proximal extremities, facial involvement is relatively uncommon and often poses a diagnostic challenge due to its clinical resemblance to more prevalent conditions, including rosacea, seborrheic dermatitis and superficial fungal

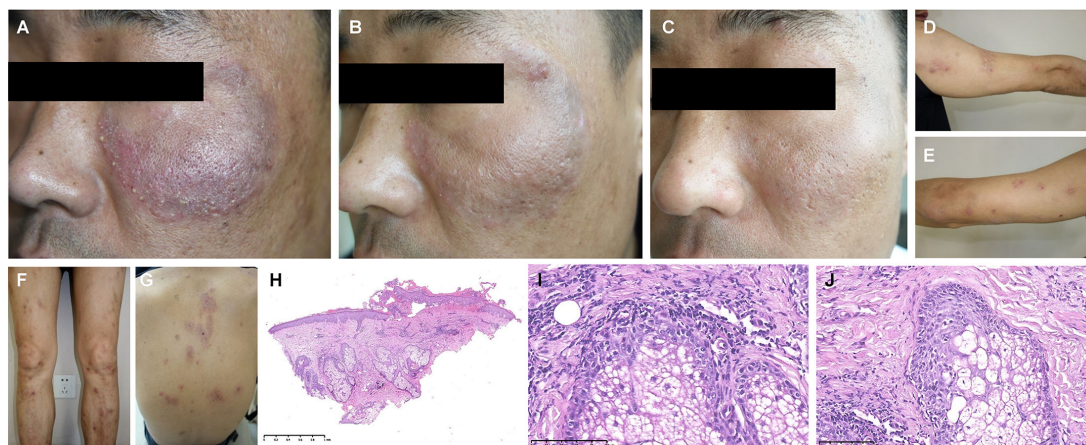


Fig. 1. (A) Clinical presentation of the facial lesion before treatment, showing an annular erythematous plaque with a raised border, scattered pustules along the margin, and central hyperpigmentation. (B, C) Marked resolution of erythema and pustules with flattening of the lesion after 1 month and 6 months of upadacitinib treatment. (D–G) Scattered erythematous papules and nodules with excoriations were observed on the trunk and extremities. (H) Histopathology showed hyperkeratosis with mild epidermal acanthosis, accompanied by dense inflammatory cell infiltration around dermal hair follicles and sebaceous glands (H&E, $\times 10$). (I, J) Infiltration of lymphocytes and eosinophils within and around folliculosebaceous units (H&E, I: $\times 100$, J: $\times 40$).

infections (4). Consequently, delayed diagnosis and inappropriate treatment are frequent in clinical practice.

Mechanistically, EPF is increasingly recognized as an inflammatory condition that may involve type 2-skewed immune responses, characterized by eosinophil recruitment, activation and persistence within the folliculosebaceous unit. Cytokines including IL-4, IL-5, and IL-13 contribute to this process through activation of the JAK–STAT signalling pathway (1). IL-5 primarily regulates eosinophil differentiation and survival via JAK2-dependent signalling, whereas IL-4 and IL-13 promote Th2 polarization and amplify local inflammation through JAK1-mediated STAT6 activation.

Recent reports describing successful treatment with dupilumab, mepolizumab, and adalimumab suggest that EPF may involve heterogeneous inflammatory pathways extending beyond eosinophilic infiltration alone (5). Dupilumab inhibits IL-4R α -mediated IL-4/IL-13 signalling (6, 7), whereas mepolizumab selectively targets IL-5-driven eosinophil activation (8, 9); adalimumab further implicates TNF- α -associated inflammatory pathways in selected patients (10). Collectively, these findings support the concept that EPF is characterized by cytokine redundancy and complex immune dysregulation rather than dependence on a single inflammatory axis.

In this context, JAK1 represents an important, but not exclusive, mediator linking epithelial–immune cross-talk and contributing to inflammatory signalling in EPF. Consistent with this mechanistic framework, emerging evidence suggests that JAK inhibition may be beneficial in refractory EPF. Clinical responses have been reported with both pan-JAK inhibitors such as tofacitinib and selective JAK1 inhibitors such as abrocitinib, although current evidence remains limited to case reports (3, 11, 12). However, most available evidence involves

pan-JAK inhibitors or limited cases treated with other agents, and the role of selective JAK1 inhibition remains insufficiently defined.

Upadacitinib, a JAK1-preferential inhibitor with partial activity against JAK2, may offer distinct therapeutic advantages in this setting (13). Compared with broader JAK inhibition (e.g., tofacitinib), its relative selectivity for JAK1 allows more targeted modulation of cytokine pathways implicated in type 2 inflammation, while potentially reducing off-target effects associated with JAK2 and JAK3 inhibition. In contrast to abrocitinib, upadacitinib may exhibit subtle pharmacologic differences. At therapeutic exposures, it demonstrates a high degree of JAK1 selectivity together with measurable activity against JAK2, which may provide complementary modulation of IL-5-mediated eosinophil biology, a central feature of EPF. Furthermore, its rapid onset of action, as observed in other type 2 inflammatory dermatoses such as atopic dermatitis, may facilitate early control of inflammation and pruritus. In our case, a rapid and marked clinical response was observed, further supporting the therapeutic potential of upadacitinib in EPF.

Nevertheless, the long-term efficacy and safety of upadacitinib in EPF remain unclear, and larger studies are required to determine optimal treatment strategies and identify patients most likely to benefit from JAK1 inhibition.

In summary, we report a rare case of EPF with prominent facial involvement that was initially misdiagnosed and refractory to conventional therapies but showed a rapid and marked response to upadacitinib. This case underscores the importance of recognizing EPF in atypical facial dermatoses and supports the emerging role of JAK1 inhibition as a

targeted therapeutic strategy. Among available agents, upadacitinib may represent a rational and potentially advantageous option, particularly in refractory cases requiring rapid disease control.

ACKNOWLEDGEMENTS

This work was supported by the Fundamental Research Funds for the Central Universities, Peking Union Medical College (grant number 3332025007) and National Natural Science Foundation of China (82573978).

Data Availability: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethical approval: This study was conducted in accordance with the principles of the Declaration of Helsinki. The authors certify that they have obtained all appropriate patient consent forms. The patient has given written consent for clinical information and images to be reported.

The authors have no conflicts of interest to declare.

REFERENCES

- Long H, Zhang G, Wang L, Lu Q. Eosinophilic skin diseases: a comprehensive review. *Clinic Rev Allerg Immunol* 2016; 50: 189–213. <https://doi.org/10.1007/s12016-015-8485-8>
- Li Y, Yang X, Lu J, Chen K, Wu W, Zhang M, et al. A case of eosinophilic pustular folliculitis misdiagnosed as eczema. *Clin Cosmet Investig Dermatol* 2024; 17: 199–204. <https://doi.org/10.2147/CCID.S445760>
- Wang QX, He HY, Niu YL, Fang S. Refractory eosinophilic pustular folliculitis treated with tofacitinib: a case series and literature review. *Clin Exp Dermatol* 2025; 50: 1196–1200. <https://doi.org/10.1093/ced/llaf053>
- Li Y, Chen G, Zhou X, Zheng X, Zhang M, Yao X, et al. Clinical and pathological analysis of 10 cases of eosinophilic pustular folliculitis. *Clin Cosmet Investig Dermatol* 2023; 16: 2467–2472. <https://doi.org/10.2147/CCID.S427718>
- Katoh M, Nomura T, Miyachi Y, Kabashima K. Eosinophilic pustular folliculitis: a review of the Japanese published works. *J Dermatol* 2013; 40: 15–20. <https://doi.org/10.1111/1346-8138.12008>
- Mansilla-Polo M, Navarro-Blanco F, López-Davía J, Botella-Estrada R. Successful treatment of recalcitrant eosinophilic pustular folliculitis with dupilumab. *Sultan Qaboos Univ Med J* 2025; 25: 622–627. <https://doi.org/10.18295/2075-0528.2873>
- Tan X, Nie S, Chen B, Wu Z. Successful treatment of eosinophilic pustular folliculitis with dupilumab in a 9-year-old boy. *Pediatr Dermatol* 2025; 42: 1044–1046. <https://doi.org/10.1111/pde.15908>
- Mansaray L, Comoz F, Domp Martin A, Kottler D. Atypical eosinophilic pustular folliculitis successfully treated with mepolizumab. *Ann Dermatol Venereol* 2026; 153: 103454. <https://doi.org/10.1016/j.annder.2025.103454>
- Chersi F, Zelin E, Mazzeletti V, Caro Caposiena DR, Hasa Z, Nan K, et al. Mepolizumab therapy for eosinophilic pustular folliculitis (Ofuji disease). *J Eur Acad Dermatol Venereol* 2024; 38: e993–e996. <https://doi.org/10.1111/jdv.20047>
- Ramondetta A, Giuffrida G, Di Vincenzo S, De Pasquale R. A case of eosinophilic pustular folliculitis successfully treated with adalimumab. *Dermatol Ther* 2022; 35: e15550. <https://doi.org/10.1111/dth.15550>
- Cai L, Yan Y, Li Y, Lin J, She X, Wang X. Two cases of eosinophilic pustular folliculitis successfully treated with abrocitinib. *J Dermatol* 2024; 51: 1694–1697. <https://doi.org/10.1111/1346-8138.17284>
- Zhu D, Lin Y, Li Y, Yao L. Tofacitinib for the management of refractory eosinophilic pustular folliculitis: a case report and literature review. *Clin Cosmet Investig Dermatol* 2026; 19: 585317. <https://doi.org/10.2147/CCID.S585317>
- Chen M, Dai SM. A novel treatment for psoriatic arthritis: Janus kinase inhibitors. *Chin Med J* 2020; 133: 959–967. <https://doi.org/10.1097/CM9.0000000000000711>