Received: 4 November 2021 | Revised: 10 January 2022 | Accepted: 19 January 2022

DOI: 10.1002/cia2.12228

Journal of Cutaneous Immunology and Allergy

WILEY

CORRESPONDENCE

Acneiform eruption during peficitinib treatment

To the Editor,

The surface of the skin maintains homeostasis, which is essential for the physiology of the skin to drive appropriate responses to external environmental factors.[1] However, once the physiological function was destroyed by environmental stimuli, decreased skin function leads to the skin diseases.[1] Drug eruption is one of the triggers to cause allergic cutaneous immune reactions to medications. On the contrary, unusual skin clinical manifestation due to medications sometimes is observed by novel medications, which help to get a better understanding of the factor-mediated skin physiological function. Herein, we report a case of acneiform eruption following a JAK inhibitor, peficitinib.

A 63-year-old male patient had been suffered from rheumatoid arthritis, and peficitinib was administrated to relieve his arthritis symptom. However, he recognized erythematous papules with pustules on his back and chest and was referred to the dermatology department for the evaluation of his skin eruption. On physical examination, erythematous papules with pustule were observed on the back and chest (Figure 1A,B). We did not conduct bacterial culture examination for

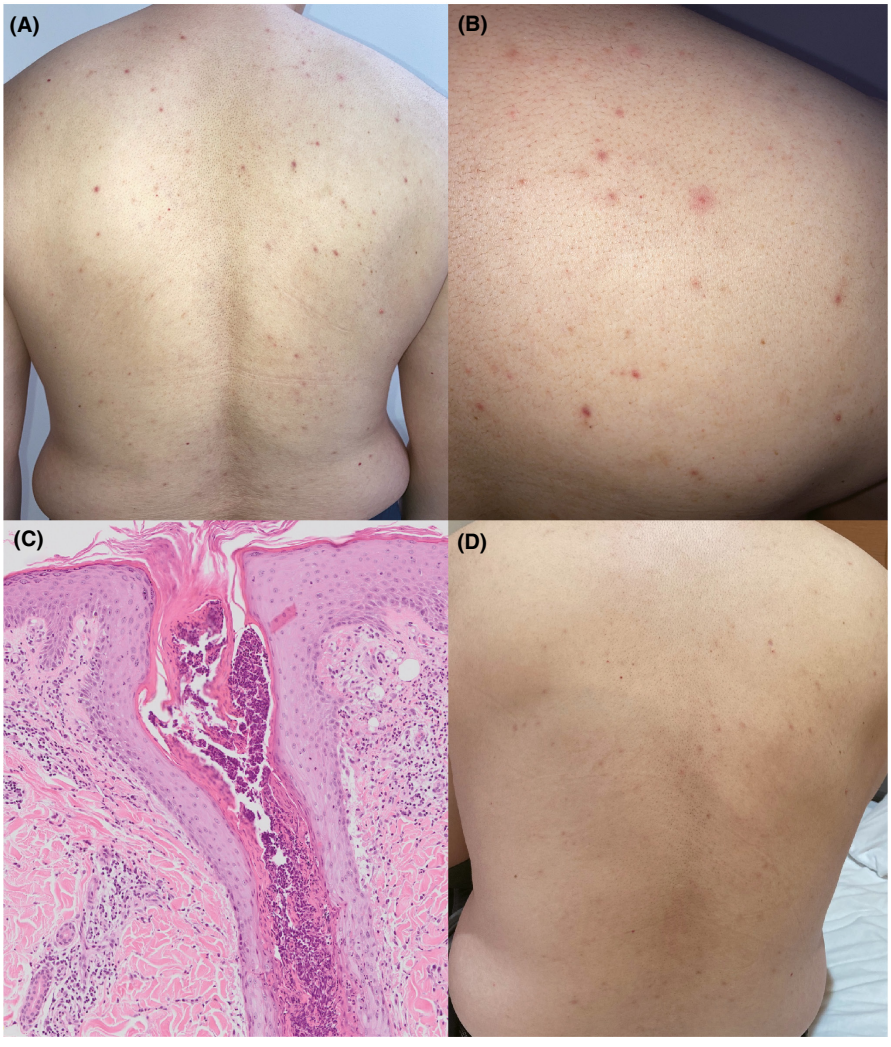

FIGURE 1 Clinical manifestation and histological examination. (A, B) Clinical manifestation during peficitinib treatment. Erythematous papules with pustule were observed in his back. (C) Histological examination. A skin biopsy taken from his back showed hair follicular keratinization with inflammatory cell infiltration around hair follicle. (D) Improved clinical manifestation after topical corticosteroid treatment

this acneiform eruption. At first, we started to treat his skin eruption by topical benzoyl peroxide as traditional acne vulgaris treatment; however, his acneiform eruption did not respond to the treatment. A skin biopsy taken from his back showed parakeratosis and hyperkeratosis on the top of the hair follicle with inflammatory cell infiltration (Figure 1C). Because his acneiform eruption was intractable by the treatment, we tried to apply topical steroids of betamethasone butyrate propionate on his acneiform eruption, which gradually improved within one month under the continuation of peficitinib administration (Figure 1D).

JAK inhibitor sometimes causes acneiform eruption; however, the detailed mechanism and therapeutic option remain controversial. Our case presented the hair follicle occlusion, and topical steroid is effective; however, the detailed mechanism remains unclear. At least, follicular keratinization played some role as one of the mechanisms of acneiform eruption in this patient. Acne is initiated following increased proliferation of keratinocytes in hair follicle infundibulum and enhanced abnormal keratinization,[2] which is regulated by epidermal growth factor in some parts.[3] JAK-STAT signaling also involves the down-stream signal transduction of the epidermal growth factor receptor.[4] Consistently, EGFR inhibitor cause acneiform eruption as the cutaneous adverse reaction. Although not all patients, but some patients might show that JAK inhibitor plays inhibitory action in the development of follicular keratinization. On the contrary, JAK inhibitors also have an opposite effect to suppress keratinocyte proliferation showing the efficacy against inflammatory skin diseases in addition to the enhancement of epidermal barrier function.[5] Therefore, there might be a heterogeneity in the distribution of JAK-related regulation of EGFR signaling involved in the acneiform eruption.

As the treatment of this case, a topical steroid is effective for his acneiform eruption following JAK inhibitor treatment; however, it should be kept in mind that therapeutic options should depend on their clinical manifestation of acneiform eruption during JAK inhibitor treatment.

DECLARATION SECTION

Approval of the research protocol: No.
Informed Consent: N/A.
Registry and the Registration No. of the study/trial: N/A.
Animal Studies: N/A.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

Reiko Hara MD
Yu Sawada MD, PhD
Motonobu Nakamura MD, PhD

Department of Dermatology, University of Occupational and Environmental Health, Kitakyushu, Japan

Correspondence
Yu Sawada, MD, PhD, Department of Dermatology, University of Occupational and Environmental Health, 1-1, Iseigaoka, Yahatanishi-Ku, Kitakyushu, Fukuoka 807-8555, Japan.
Email: long-ago@med.uoeh-u.ac.jp

ORCID

Yu Sawada https://orcid.org/0000-0001-8793-708X

REFERENCES

1. Sawada Y, Nakatsuji T, Dokoshi T, Kulkarni NN, Liggins MC, Sen G, et al. Cutaneous innate immune tolerance is mediated by epigenetic control of MAP2K3 by HDAC8/9. Sci Immunol. 2021;6(59):eabe1935.
2. Schneider MR, Paus R. Deciphering the functions of the hair follicle infundibulum in skin physiology and disease. Cell Tissue Res. 2014;358(3):697–704.
3. Frew JW. Hidradenitis suppurativa is an autoinflammatory keratinization disease: a review of the clinical, histologic, and molecular evidence. JAAD Int. 2020;1(1):62–72.
4. David M, Wong L, Flavell R, Thompson SA, Wells A, Larner AC, et al. STAT activation by epidermal growth factor (EGF) and amphiregulin. Requirement for the EGF receptor kinase but not for tyrosine phosphorylation sites or JAK1. J Biol Chem. 1996;271(16):9185–8.
5. Amano W, Nakajima S, Kunugi H, Numata Y, Kitoh A, Egawa G, et al. The Janus kinase inhibitor JTE-052 improves skin barrier function through suppressing signal transducer and activator of transcription 3 signaling. J Allergy Clin Immunol. 2015;136(3):667–77.e7.

How to cite this article: Hara R, Sawada Y, Nakamura M. Acneiform eruption during peficitinib treatment. J Cutan Immunol Allergy. 2022;5:139–140. doi:10.1002/cia2.12228