

Letter to the Editor

Tofacitinib-induced acne in a patient of vitiligo vulgaris: A case report

Anisha Biswal¹, Abhipsa Samal¹, Maitreyee Panda¹

¹Department of Dermatology, Institute of Medical Sciences and SUM Hospital, Bhubaneswar, Odisha, India.



***Corresponding author:**

Anisha Biswal,
Department of Dermatology,
Institute of Medical
Sciences and SUM Hospital,
Bhubaneswar, Odisha, India.
richirichbiswal@gmail.com

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Dear Sir,

The recent advances in the understanding of the pathogenesis of several dermatologic diseases have led to emergence of new targeted therapeutic molecules. One such molecule is Janus Kinase (JAK) inhibitors which are a promising group of small molecules in the treatment of immune mediated diseases. There are few cutaneous adverse effects that are reported involving the use of JAK inhibitors which includes acne, eczema herpeticum, and herpes zoster.^[1] Acne has been reported, commonly in atopic dermatitis and rarely in other populations such as rheumatoid arthritis, psoriatic arthritis, spondyloarthritis, inflammatory bowel disease, and vitiligo.^[2] Upadacitinib-induced acne in atopic dermatitis has been recently reported in an randomized control trial.^[3] Tofacitinib-induced acne exacerbation has also been reported in a patient of alopecia areata.^[4] We hereby report a case of tofacitinib-induced acne in an adolescent girl with vitiligo vulgaris.

A 17-year-old average built female presented to the dermatology outpatient department with multiple erythematous papules, pustules and comedones on the face for 3 weeks [Figure 1].



Figure 1: Multiple erythematous papules, pustules, and comedones over the entire face post intake of tofacitinib.

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There was no history of prior acne vulgaris, menstrual irregularities, or application of any topical corticosteroids. She was a known case of progressive vitiligo for 5 years with lesions on the chest and bilateral lower legs [Figure 2]. She had been on multiple treatments previously for the same without any significant improvement. After all baseline investigations, she was started on oral tofacitinib 5 mg twice daily. Within 1 month of tofacitinib intake, she developed the acne lesions over the entire face. On examination, there were multiple erythematous papules, pustules, and predominantly comedones over bilateral cheek and forehead indicating Grade 2 acne. There was no truncal involvement. Acne severity was assessed using global acne severity grading system. Oral tofacitinib was temporarily withdrawn for 15 days and the patient was treated for Grade 2 acne with oral doxycycline 100 mg twice daily along with topical adapalene. There was significant improvement of the acne lesions within 15 days. When tofacitinib was restarted again after a gap of 4 weeks, new acne lesions started reappearing which indicates the temporal association between tofacitinib and appearance of acne.

JAK inhibitors has proven its efficacy in various inflammatory dermatoses as off label indication including vitiligo. JAK inhibitors inhibits the activity of JAK enzymes that interfere with the pathway of intracellular signalling, which is associated with various cutaneous and systemic side effects. Some of the common side effects being reactivation of infections (hepatitis, tuberculosis, and upper respiratory tract infections) anemia, thrombocytopenia, neutropenia, dearranged lipid profile, acneiform eruptions, and acne vulgaris.

The detailed mechanism of JAK inhibitor-related to acne remains unclear. Acne is usually caused by hyperkeratinization and obstruction of sebaceous follicles resulting from abnormal keratinization of infundibular epithelium which is partly regulated by the epidermal growth factor. JAK-STAT signaling involves the downstream signal transduction of epidermal growth



Figure 2: Development of acne vulgaris after tofacitinib intake for vitiligo patches over bilateral lower legs.

factors which leads to hyperkeratinization of follicles.^[5] Another possibility is that inflammatory lesions may be induced by an immune slant toward T helper 1 and 17 after T helper 2 signaling inhibition. Apart from this, JAK inhibitors cause immune inhibition and have an important role in the change of microbial colonization of the skin, including the colonization of demodex folliculorum.^[6] Contradictorily, a case-control study suggests overexpression of JAK 1 and JAK 3 in acne skin lesions, compared to non-lesional skin indicating activation of JAK signaling pathway in acne, which can be used as a therapeutic modality in the treatment of acne.^[7] There is still a marked diversity in the correlation between the JAK pathway and acne. Limited cases have been reported pertaining to side effects of tofacitinib till date implying the need of further studies to assess the role of JAK signaling in the expression of acne.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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