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Sequential paradoxical inflammatory dermatoses in hidradenitis suppurativa following TNF-α and IL-17 pathway inhibition

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Introduction

Paradoxical inflammatory reactions are increasingly recognised adverse events of biologic therapy and represent a significant clinical challenge in the management of immune-mediated dermatoses. Hidradenitis suppurativa (HS), characterised by complex cytokine dysregulation involving TNF-α, IL-17 and innate immune pathways, may predispose patients to such reactions[1].

Materials and Methods

We report the case of a 46-year-old woman with HS diagnosed in 2013, who previously achieved excellent disease control with oral clindamycin and rifampicin. Approximately nine years later, adalimumab was initiated for disease recurrence. Shortly after treatment commencement, she developed rapid and severe clinical deterioration with inflammatory nodules, abscesses and ulcerated lesions consistent with HS flare in areas previosuly unaffected by HS. In the absence of infection, immunogenicity or secondary loss of response, this presentation was considered paradoxical HS induced by TNF-α inhibition[1]. The flare responded promptly to systemic prednisolone and amoxicillin–clavulanate and adalimumab was discontinued.

The patient was subsequently transitioned to secukinumab in January 2025 with a concomitant corticosteroid taper and later escalated to bimekizumab 3 months later, due to persistent disease activity. After approximately six months of IL-17A/F inhibition, she developed new-onset, well-demarcated erythematous scaly plaques clinically consistent with paradoxical psoriasis, without any personal or family history of psoriasis[3]. This was followed in early November 2025 by the onset of painful, tender subcutaneous nodules on the lower limbs, with histopathology confirming erythema nodosum.

Results

Mechanistically, TNF-α blockade may induce paradoxical inflammation through unchecked activation of plasmacytoid dendritic cells and increased type I interferon signalling, leading to aberrant cutaneous immune responses[2]. Similarly, inhibition of the IL-17 pathway may disrupt cytokine homeostasis, with compensatory activation of alternative inflammatory circuits, potentially contributing to multisystem inflammatory manifestations including psoriasiform disease and panniculitis[2,3]. This case is notable for the occurrence of sequential and overlapping paradoxical inflammatory dermatoses across different biologic classes in a single HS patient.

Conclusions

Recognition of paradoxical reactions is essential to avoid misdiagnosis as disease progression or therapeutic failure. This

case highlights the importance of immunopathogenic understanding when selecting and sequencing biologic therapies in HS.