Dermatology Reports
https://www.pagepress.org/journals/index.php/dr/index

eISSN 2036-7406

Please cite this article as:

Sbarra G, Mortato E, Loconsole F. Upadacitinib for the management of refractory hidradenitis suppurativa, psoriasis, and pyoderma gangrenosum. *Dermatol Rep 2026 [Epub Ahead of Print] doi: 10.4081/dr.2026.10314*

Submitted 23/02/25 - Accepted 28/01/26

Upadacitinib for the management of refractory hidradenitis suppurativa, psoriasis, and pyoderma gangrenosum

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Key words: psoriasis; hidradenitis suppurativa; pyoderma gangrenosum; upadacitinib.

Contributions: Edoardo Mortato and Giorgia Sbarra: conceptualization; methodology; data curation; formal analysis; investigation; writing – original draft; Francesco Loconsole: supervision; writing – review & editing. All authors have read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of interest: the authors have no conflict of interest to declare.

Ethics approval and consent to participate: not required.

Consent for publication: the patient in this manuscript has given written informed consent to the publication of her case details and any accompanying images.

Availability of data and materials: data supporting the findings of this study are available on request from the corresponding author.

Dear Editor,

Hidradenitis suppurativa (HS) is a chronic immune-mediated skin disease presenting with recurrent inflammatory nodules, abscesses, draining tunnels, and scarring, most commonly in intertriginous sites. Its estimated global prevalence is approximately 1% and its pathogenesis is complex, involving dysregulation of several pro-inflammatory cytokines, including tumor necrosis factor (TNF)-α and interleukin (IL)-17.[1,2] HS frequently coexists with systemic inflammatory conditions, such as axial spondyloarthropathies and inflammatory bowel diseases (IBD).[3,4] Additional associations with dermatologic diseases have been described, particularly with pyoderma gangrenosum (PG). Emerging evidence also suggests a potential link between HS and psoriasis, possibly due to overlapping cytokine pathways.[5,6] When these conditions occur together, management becomes particularly challenging and may require therapeutic strategies targeting broad inflammatory networks.

We report the case of a 29-year-old woman with a long-standing history of HS and psoriasis, who also suffered from ulcerative colitis complicated by episodes of PG. Her medical history included Stickler syndrome, a connective tissue disorder without known association with HS or psoriasis.[7] She presented in October 2023 with a severe flare of HS involving axillae, buttocks, breasts, abdomen, groin, and retroauricular areas, accompanied by infiltrated erythematous plaques consistent with inverse psoriasis. PG was also active at the time of evaluation (Figure 1). Pain was severe and accompanied by recurrent fever. Her baseline disease burden included a Psoriasis Area Severity Index (PASI) of 6, a Dermatology Life Quality Index (DLQI) of 28, a Numeric Pain Rating Scale (NPRS) score of 9, and a Sartorius score of 83.5.

The patient had previously been treated with adalimumab, which was discontinued due to inadequate control of gastrointestinal symptoms. Given the worsening of both intestinal and cutaneous disease, a multidisciplinary decision with gastroenterology was made to initiate upadacitinib 45 mg/day, following the approved dosing regimen for ulcerative colitis.

Upadacitinib is a selective Janus kinase 1 (JAK1) inhibitor that modulates the JAK-signal transducer and activator of transcription (STAT) signaling pathway and reduces the activity of cytokines such as IL-6, IL-17, IL-22, IL-23, and interferon (IFN)-γ.[8,9] Targeting this pathway is biologically plausible in HS, where dysregulation of TNF-α, IFN-γ, and the IL-12/IL-23 axis is well documented.[10,11]

After 16 weeks of therapy at 45 mg/day, the patient achieved significant improvement across all disease domains. Her DLQI decreased from 28 to 5, NPRS from 9 to 0, and Sartorius score from 83.5

to 27.5. Psoriasis and PG showed marked remission. The improvement was not limited to inflammatory burden but also reflected better quality of life and pain control.

In February 2024, the dose of upadacitinib was reduced to 30 mg/day, consistent with maintenance dosing in IBD. However, within one month, she experienced a partial relapse of psoriasis and recurrence of painful axillary nodules, with an NPRS of 7, DLQI of 24, PASI of 3.6, and a Sartorius score of 50.5 (Figure 2); PG did not recur. Although disease activity worsened compared with the end of the 45 mg phase, severity remained lower than baseline. The relapse was milder, limited to fewer sites, and lacked systemic manifestations, suggesting a dose-dependent response.

This case highlights several clinically relevant considerations. First, upadacitinib 45 mg/day demonstrated substantial benefit in a patient with severe multi-system inflammatory disease refractory to anti-TNF therapy. The dramatic improvement in HS, psoriasis, and PG supports the hypothesis that JAK1 inhibition may act on shared inflammatory pathways involved in these conditions. Second, the partial relapse following dose reduction suggests that some patients with severe HS may require sustained higher doses for optimal disease control. Establishing an evidence-based dosing strategy for HS will require controlled studies, as current data remain limited. Third, although the patient had Stickler syndrome, no evidence in the literature supports an association with inflammatory skin diseases. Genes implicated in Stickler syndrome have not been linked to HS or psoriasis, suggesting the coexistence in this patient is coincidental.

Finally, recent genetic studies have identified shared susceptibility loci between HS and psoriasis, characterizing a distinct subgroup with increased morbidity and elevated risk for Crohn's disease.[12] Recognizing this phenotype may refine clinical management and guide therapeutic decisions, particularly in patients with complex multimorbidity similar to our case.

Given the encouraging clinical response observed in this patient, further research – including prospective studies and randomized trials – is warranted to evaluate the efficacy, safety, and long-term outcomes of upadacitinib in HS, particularly in patients with concurrent immune-mediated diseases. JAK1 inhibition may represent a promising therapeutic approach for individuals with overlapping inflammatory conditions requiring broad cytokine pathway modulation.

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Figure 1. Patient's clinical condition at baseline: evident purulent nodular and plaque lesions in the left axilla (**a**), in the right submammary region (**b**), and plaques localized in the retroauricular region (**c**), along with an associated ulcerative lesion on the right thigh (**d**).

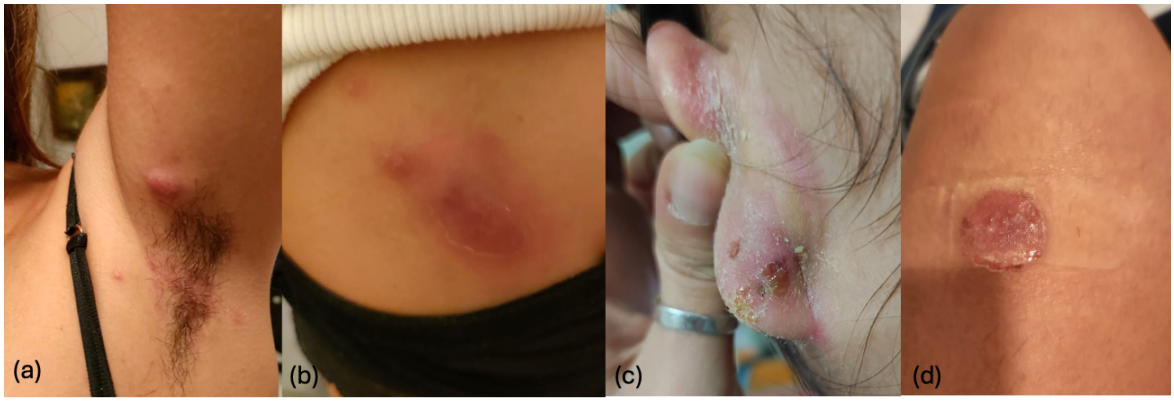

Figure 2. Relapse of hidradenitis suppurativa following the reduction of upadacitinib to 30 mg daily. The recurrence is localized primarily to the axillary area (**a**, **b**), with no clinical involvement of the breasts or other previously affected sites. Ulcerative lesion on the right thigh has resolved (**c**).