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When anti-tumor necrosis factor-α backfires: paradoxical psoriasis and psoriatic arthritis in a patient with hidradenitis suppurativa

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Dear Editor,

Hidradenitis suppurativa (HS) is a chronic inflammatory disease of the folliculoapocrine units.[1,2] Biological therapies, historically led by the anti-tumor necrosis factor (TNF)-α antibody adalimumab, have transformed severe HS management.[3] Recently, interleukin (IL)-17 inhibitors (secukinumab, bimekizumab) were also approved in Europe for moderate-to-severe HS. However, TNF-α blockade can paradoxically induce or exacerbate inflammatory skin and joint conditions, including psoriasis (incidence 1-5%) and psoriatic arthritis.[4-7] We report a case of paradoxical inverse psoriasis and suspected early psoriatic arthritis induced by adalimumab in an HS patient, successfully managed by switching to secukinumab.

A 26-year-old female smoker (body mass index 26 kg/m^2) with no personal or family history of psoriasis or autoimmunity presented with severe HS (Hurley stage III). Previous antibiotic courses provided temporary relief. Baseline inflammatory activity was high (International Hidradenitis Suppurativa Severity Score System [IHS4]=34: presenting with 4 inflammatory nodules, 2 abscesses, and 6 draining tunnels). Standard adalimumab therapy was initiated (Figure 1).

After 16 weeks, inflammatory lesions improved significantly (IHS4=6: 2 nodules, 0 abscesses, 1 tunnel), although the structural Hurley stage III persisted due to irreversible scarring. At month 6, new erythematous, well-demarcated scaling plaques appeared in the inguinal and axillary folds and on the scalp. Intertriginous infections were excluded via negative mycological and bacterial cultures. Disease severity was assessed using the static Physician's Global Assessment of Genitalia (sPGA-G), which scored 3, and the Psoriasis Scalp Severity Index (PSSI), which scored 38 (Figure 2).

Simultaneously, the patient developed distal interphalangeal and sacroiliac joint pain. Ultrasound revealed tendon calcifications and early synovitis. C-reactive protein (CRP) was 9.5 mg/L; rheumatoid factor and anti-cyclic citrullinated peptide were negative. Although the Classification Criteria for Psoriatic Arthritis (CASPAR) were incomplete, inflammatory arthralgia and suspected early psoriatic arthritis were diagnosed. Given the temporal onset, dechallenge effect, and absence of prior history, these manifestations were possibly adalimumab-induced, though a *de novo* unmasking of disease associated with HS remains an alternative explanation.

Despite sustained HS benefit, adalimumab was discontinued. Secukinumab was initiated (300 mg weekly for 5 weeks, then 300 mg every 4 weeks). At week 24, psoriatic lesions showed marked improvement (sPGA-G=0; residual scalp scaling PSSI=7) (Figure 3). HS inflammatory remission continued (IHS4=4: 0 nodules, 0 abscesses, 1 tunnel). CRP normalized (1.5 mg/L), with complete resolution of joint symptoms. No adverse events occurred during a 12-month follow-up.

Paradoxical reactions to anti-TNF-α agents challenge the management of chronic inflammatory diseases.[8] The proposed mechanism involves TNF-α blockade dysregulating the type I interferon (IFN)/IL-23/IL-17 axis, causing excessive IFN-α production by plasmacytoid dendritic cells and subsequent Th17 activation.[9,10]

In our patient, inverse psoriasis and suspected early psoriatic arthritis developed after six months of adalimumab. Intertriginous and scalp localizations frequently indicate an increased risk of joint involvement in psoriatic disease.[11] Discontinuation of adalimumab and switching to secukinumab, a selective IL-17A inhibitor, yielded rapid clinical improvement. Secukinumab is efficacious in psoriasis, psoriatic arthritis, and moderate-to-severe HS.[12] However, the relationship between secukinumab and HS is complex; paradoxical HS has also been reported with IL-17 blockade, indicating biologics can act as "friends or foes", depending on the immunological context.[8,13]

This case underscores the importance of recognizing paradoxical reactions and selecting an alternative target. While hypothesis-generating, switching to an IL-17A inhibitor provided simultaneous control of HS, psoriasis, and inflammatory arthralgia, highlighting its potential utility in complex phenotypes.

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Figure 1. Clinical presentation of severe hidradenitis suppurativa (Hurley stage III) at baseline prior to adalimumab initiation. **A**) Inguinal and **B**) axillary regions showing active inflammatory nodules and draining tunnels.

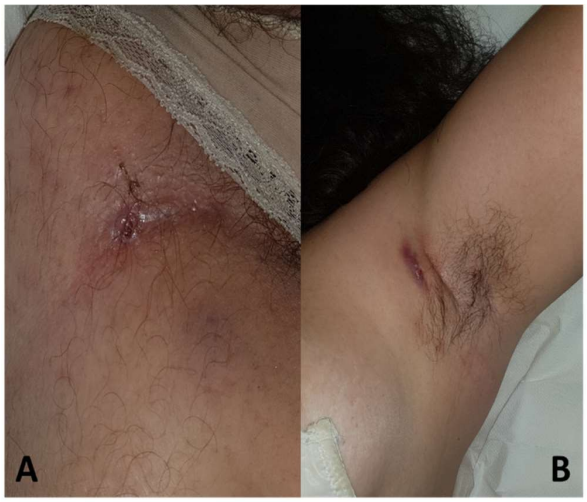

Figure 2. Clinical onset of paradoxical psoriasis after 6 months of adalimumab therapy. **A**) Pubic and **B**) anal regions displaying well-demarcated erythematous plaques typical of inverse psoriasis. **C**) Scalp region exhibiting thick, adherent, scaling plaques.

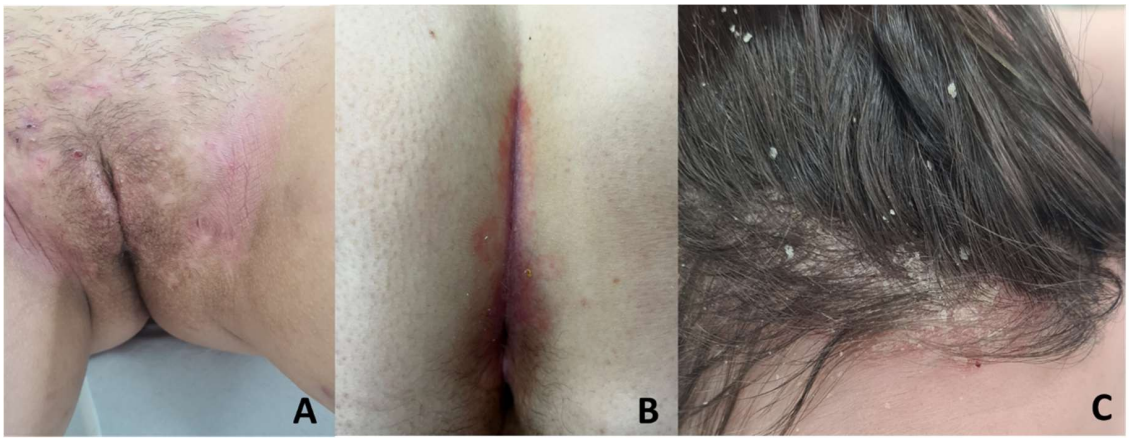

Figure 3. Clinical response after 24 weeks of secukinumab therapy. **A**) Pubic and **B**) anal regions showing complete clearance of the psoriatic plaques. **C**) Scalp region demonstrating marked improvement with only mild residual scaling, consistent with the reported PSSI score of 7.