Inverse psoriasis on pre-existing hidradenitis suppurativa lesions: Reply to "A case series of tumor necrosis factor inhibitor-induced psoriasis in patients with hidradenitis suppurativa"

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To the Editor:

We read "A case series of tumor necrosis factor inhibitor-induced psoriasis in patients with hidradenitis suppurativa" with great interest [1]. We recently encountered a case of inverse psoriasis that developed on long-standing hidradenitis suppurativa (HS) lesions. The patient, a 41-year-old man, presented with acute onset erythema in the axillary regions and reported suffering from severe, chronic HS in the axilla and inguinal regions for nearly 20 years. The patient was a non-smoker, and his body mass index was 30.7kg/m^2 (normal range: 18.5–24.9kg/m^2). His medical history was otherwise insignificant. The patient's sister had psoriasis localized to scalp. He had undergone multiple surgeries previously and was currently being treated with doxycycline. He had not used any topical treatments on these newly-formed lesions. On physical examination, the patient had inactive Hurley stage III lesions in both axilla and inguinal areas and large erythematous plaques were observed in the right axillary region (**Figure 1A**, **B**). Erythematous plaques were also present on the neck, umbilicus, and left eyelid.

A biopsy from the axilla revealed hyperkeratosis and parakeratosis in epidermis and neutrophilic infiltration in parakeratotic areas (**Figure 2**). A periodic acid-Schiff stain and KOH examination were also done to exclude a possible tinea infection and they were negative. Correlation of the clinical lesions, pathology, and response to therapy was suggestive of psoriasis and based on location, inverse psoriasis. The patient was prescribed methylprednisolone cream once daily. The lesions regressed with this regimen within two months of topical corticosteroid initiation, but after six months the patient had a severe flare up in HS lesions and adalimumab was initiated in typical HS dosage. Currently the patient is

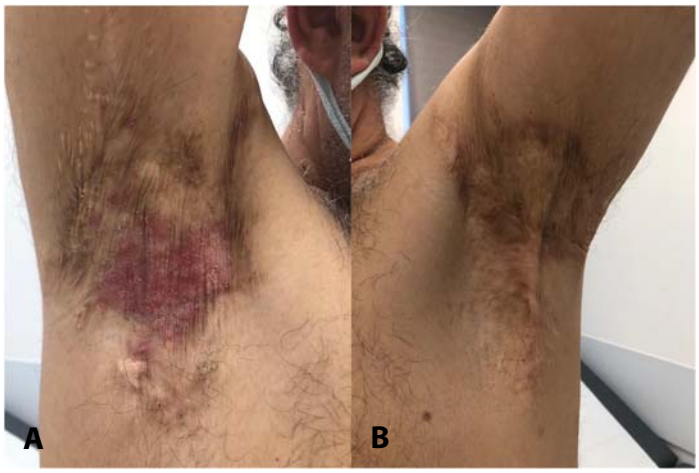

Figure 1. *Clinical image of patient showing erythematous plaques on **A)** right, and **B)** left axilla on pre-existing surgical scars from hidradenitis suppurativa lesions.*

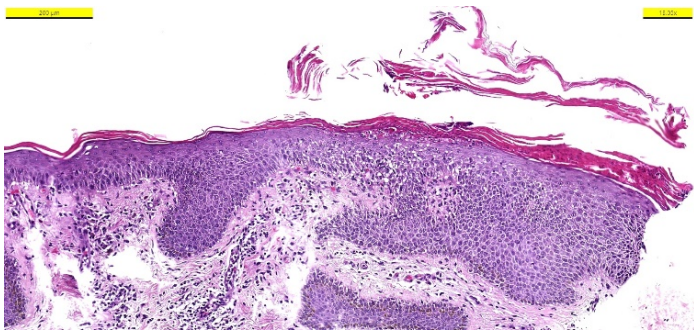

Figure 2. *Biopsy from right axilla showing prominent hyperkeratosis, foci of parakeratosis, and collection of neutrophils at the upper part of epidermis, forming small spongiform pustule. H&E, 13×.*

in remission for both diseases under adalimumab therapy (**Figure 3**).

Shared mechanisms of pathogenesis in HS and psoriasis may potentiate the development of secondary psoriasis in HS patients treated with TNF inhibitors. Although Kelly et al. have reported psoriasis as a complication of TNF inhibitor treatment in patients with HS [1], our patient had only received antibiotics for HS and had never been on immunologic treatment. Despite this, the patient developed psoriatic plaques on pre-existing HS lesions. The development of psoriasis on preexisting HS lesions in our patient could be explained by the immune compromised district (ICD) theory. There is current literature that discusses the development of squamous cell carcinoma on pre-existing HS lesions as a potential outcome of ICD [2]. In our case, the chronic inflammation and damage caused by HS lesions may have created an ICD, potentiating further inflammation in the affected skin, allowing psoriasis to develop over the preexisting lesions [3].

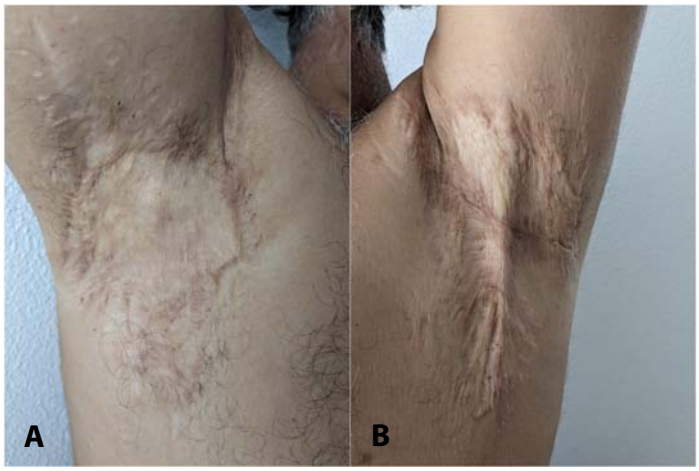

Figure 3. *Clinical image of patient showing regressed areas on **A)** right, and **B)** left axilla with pre-existing surgical scars from HS lesions.*

The cooccurrence of HS and psoriasis have been explored and reported odds ratio for HS patients of having psoriasis as compared to the background population is 2.677-2.99 [4,5]. Psoriasis and HS have a number of common factors in their disease processes, both marked by imbalances in how the innate and adaptive immune systems interact. When various cell types, such as keratinocytes, natural killer T cells, and macrophages stimulate dendritic cells, these dendritic cells produce cytokines such as IL23 and IL12. These cytokines then trigger the transformation of native T cells into Th17 and Th1, respectively. IL23 is particularly important for the survival and growth of Th17 and Th22 cells [6].

Although the demographic studies show a significant association of idiopathic HS and psoriasis, the clinical information on the type of psoriasis triggered is scarce. Kelly et al. reports six patients with psoriasis (scalp psoriasis N= 2, pustular psoriasis N=1, and plaque psoriasis N=3), [1]. Inverse psoriasis triggered with infliximab therapy in an HS patient was reported previously [7]. Further studies are needed to understand the relation between HS and psoriasis and if HS patients are more prone to develop inverse psoriasis on previous HS lesions.

Potential conflicts of interest

The authors declare no conflicts of interest.

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