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References

1. Greywal T, Zaenglein AL, Baldwin HE, Bhatia N, Chernoff KA, Del Rosso JQ, *et al.* Evidence-based recommendations for the management of acne fulminans and its variants. J Am Acad Dermatol 2017;77:109–17.

2. Marasca C, Fabbrocini G, Abategiovanni L, Camela E, Nocerino M, Di Guida A, *et al.* Adalimumab in the Management of Isotretinoin-Induced Acne Fulminans: report of a Case. Skin Appendage Disord 2021;7:115–9.

3. Legal K, Misery L. Isotretinoin-induced Acne Fulminans without Systemic Symptoms Treated Successfully with Oral Dapsone. Acta Derm Venereol 2020;100:adv00036.

4. Lages RB, Bona SH, Silva FV, Gomes AK, Campelo V. Acne fulminans successfully treated with prednisone and dapsone. An Bras Dermatol 2012;87:612–4.

5. Fakih A, Goens J, Grozdev I, Dangoisse C, Richert B. Acne fulminans induced by a low dose isotretinoin: case report and review of the literature. Dermatol Online J 2020;26:13030.

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The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

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Unveiling the bridge between neutrophilic and autoinflammatory spectrum

Good response to old but gold colchicine in a complex clinical association of Sweet Syndrome with psoriasis and hidradenitis suppurativa under treatment with ixekizumab

A 41-year-old woman sought for medical consultation to our department due to the sudden onset of fever together with painful,

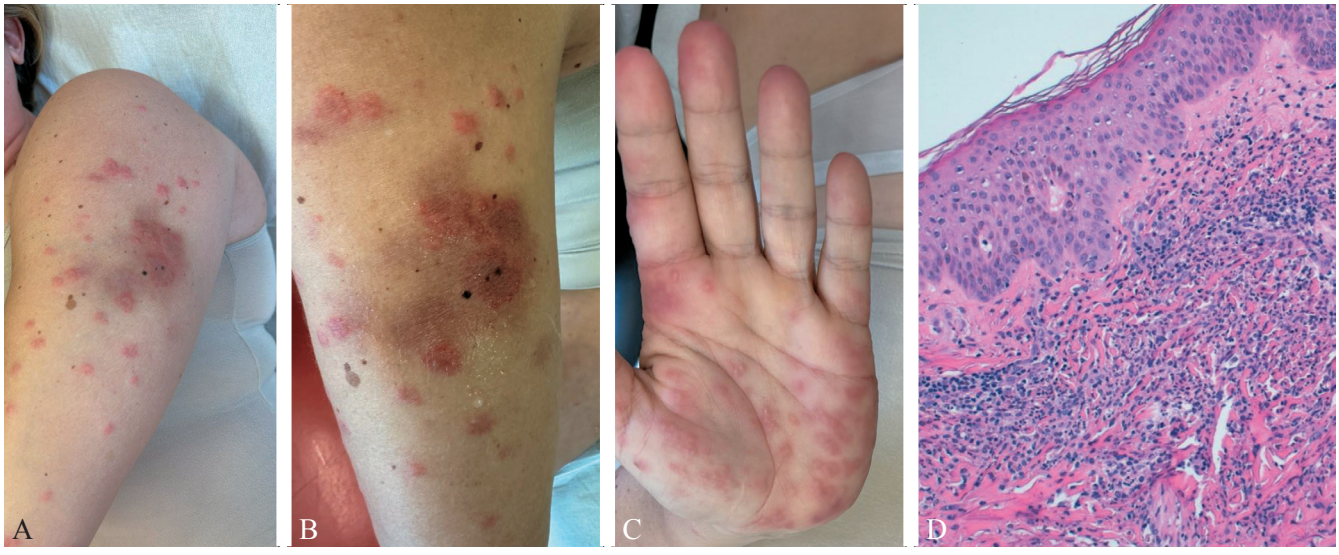

Figure 1.—Clinical photos: A) left arm is affected by painful tender erythematous lesions, with marked rim and asymmetrically distributed in a relief of mountain-range appearance; B) detail of the aforementioned lesions; C) hand palms shows little round bull's-eye target lesions, with dark red center and lighter pink edges, in an erythema multiforme-like fashion; D) a 6-mm punch biopsy revealed hyperorthokeratosis, acanthosis and focal spongiosis, mild edema of the papillary dermis, perivascular and interstitial infiltrate, composed predominately of neutrophils, with no evidence of necrotic keratinocytes or vasculitis, 20x EE.

erythematous lesions affecting asymmetrically her limbs, trunk, face and hand palms. Laboratory tests showed pharyngeal swab positive for streptococcal colonization and neutrophil leukocytosis. At clinical observation, the aforementioned lesions looked like scarlet nuance edematous plaques, sharply demarcated by an infiltrated peripheral rim in a relief of mountain-range appearance (Figure 1A-C) while hand palms showed targetoid papules in an erythema multiforme-like (EM) fashion. She suffered from hidradenitis suppurativa (HS), scalp and nail psoriasis. She was receiving ixekizumab 80 mg monthly after a first therapeutic course with adalimumab could not achieve complete psoriasis control.

Based on anamnesis and clinical appearance we raised suspicion for an outbreak of neutrophilic acute febrile dermatosis/Sweet Syndrome (SS) secondary to streptococcal infection. Consequently, we performed a 6 mm punch biopsy and histopathological findings resulted compatible with SS, with no evidence of necrotic keratinocytes or vasculitis (Figure 1D).

Precautional blood screening for autoimmune and paraneoplastic conditions was advised with no significant findings. Prednisone 0.5 mg/kg with slow tapering and amoxicillin-clavulanic acid were prescribed. The response to corticosteroid therapy was dramatic although at the beginning of tapering the patient reported a mild relapse of SS lesions. Thus, colchicine 1 mg/*die* was added as steroid-sparing agent in order to sustain and consolidate the therapeutic response, leading to steroid cessation in 3 months.

In this case report, some peculiar features can be highlighted: firstly, the occurrence of SS triggered by infectious stimulus in a patient already affected by psoriasis and HS, and secondly the dual clinical presentation of SS, marked by typical lesions on the body together with EM-like lesions at palms.

Notably, neutrophil dermatosis of the dorsal hands is regarded as a subset of SS that can involve acral areas, but in our patient dorsal hands were spared. To our knowledge, the association of psoriasis, HS and SS characterized by two coexisting phenotypes on the body and hand palms has never been reported in literature.[1] Psoriasis and HS share some pathophysiological basis, namely the activation of specific proinflammatory signaling pathways including TNF-α, IL-1, IL-12/23 and IL-17, resulting in neutrophils 'sterile' infiltration of the epidermal or dermal layer.

Owing to that a patient already affected by psoriasis and HS could be prone to develop another neutrophilic dermatosis, such as pustolar psoriasis or SS, following an appropriate inflammatory stimulus like a bacterial infection. On this regard, neutrophils' double-edge knife behavior is crucial, from one side acting as pivotal effectors of innate immunity but from the other promoting cutaneous inflammation and disrupting keratinocytes homeostasis.[2] Moreover keratinocytes can enhance neutrophil NETs formation through IL36 overproduction, in a sort of self-maintaining inflammatory loop.[3]

From a therapeutic point of view ixekizumab, which resulted effective on our patient psoriasis and HS, could not counteract her tendency to develop SS. This is an interesting point since anti-IL17 are successfully used in other neutrophils centered conditions as pustolar psoriasis, pyoderma gangrenosum and Bechet's disease, while there are scarce evidences of their effectiveness in SS. Wu *et al.* reported of a refractory SS treated with brodalumab,[4] but further studies are needed to elucidate the role of Th1/Th17 axis interplay in SS pathogenesis, and clarify if anti-IL17 drugs could represent a therapeutic choice in SS.

On the contrary, colchicine represents a remarkable option in the drug armory for SS.

In spite of affordable side effects, it boasts pleotropic pharmacodynamic actions, above all inhibition of neutrophilic extracellular traps release, which is hypothesized to be one of the leading pathogenetic mechanism either of autoinflammatory and neutrophilic disease.[5]

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References

1. Marzano AV. Hidradenitis suppurativa, neutrophilic dermatoses and autoinflammation: what's the link? Br J Dermatol 2016;174:482–3.

2. Bonnekoh H, Erpenbeck L. Neutrophilic dermatoses - Pathomechanistic concepts and therapeutic developments. J Dtsch Dermatol Ges 2023;21:374–80.

3. Nakabo S, Romo-Tena J, Kaplan MJ. Neutrophils as Drivers of Immune Dysregulation in Autoimmune Diseases with Skin Manifestations. J Invest Dermatol 2022;142(3 Pt B):823–33.

4. Wu PC, Chen CB, Chung WH, Chi CC. Brodalumab for the treatment of refractory Sweet syndrome. Int J Dermatol 2024;63:1790–1.

5. Li S, Ying S, Wang Y, Lv Y, Qiao J, Fang H. Neutrophil extracellular traps and neutrophilic dermatosis: an update review. Cell Death Discov 2024;10:18.

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Authors' contributions
Michele Lanzetti: ideation of the article, photographs achievement, drafting and revision with final approval. Filippo Toaiari: substantial contribution to ideation, drafting and revision; performed biopsy and dermoscopy examination. Emanuele M. Cipollini: main contribution to patient treatment and follow-up, ideation and critical revision. Daniela Massi, Ilaria C. Galli: contribution to drafting and revision; histopathology sampling and examination of specimen; histopathology and immunohistochemistry photographs achievement. Emiliano Antiga: main contribution to patient treatment and follow-up, ideation and critical revision, final approval.

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