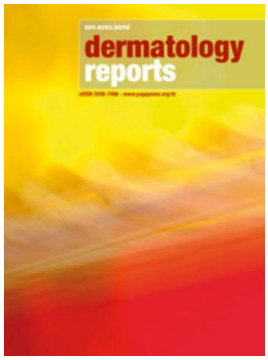

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Hidradenitis suppurativa characterized by type 2-skewed inflammatory features when associated with atopic dermatitis: critical appraisal of a dupilumab-treated case report

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Contributions: Niccolò Gori: conceptualization, data analysis and interpretation, writing - original draft; Elena Ippoliti, Flaminia Antonelli, Ludovica Manieri, Ketty Peris: data analysis and interpretation, writing - review & editing. All the authors read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of interest: NG has served as advisory board member and received honoraria for lectures for AbbVie, Sanofi and Leo Pharma; KP has served on advisory board, received honoraria for lectures and/or research grants for Abbvie, Almirall, Lilly, Galderma, Leo Pharma, Pierre Fabre, Novartis, Sanofi, Sun Pharma, Janssen. The other authors have no competing interests to declare.

Ethics approval and consent to participate: no ethical committee approval was required for this case report by the Department, because this article does not contain any studies with human participants or animals. Informed consent was obtained from the patient included in this study.

Informed consent: written informed consent for publication of clinical data and images was obtained from the enrolled patient.

Availability of data and materials: all data generated or analyzed during this study are included in this published article.

Abstract

Atopic dermatitis (AD) and hidradenitis suppurativa (HS) are two common chronic inflammatory skin diseases associated with a significant impairment of patients' quality of life. Although AD and HS are substantially different from a pathogenetic perspective, recent findings have indicated the existence of potential common pathogenetic mechanisms, including skin barrier alteration. We describe a 29-year-old male with concomitant HS and AD who obtained a marked improvement of both diseases under treatment with dupilumab, a monoclonal antibody that inhibits interleukin (IL)-4/IL-13 signaling. This finding supports the hypothesis that type 2 inflammation and skin barrier dysfunction play a significant role in the pathogenesis of HS.

Introduction

Atopic dermatitis (AD) and hidradenitis suppurativa (HS) are two chronic inflammatory skin diseases that greatly affect patients' quality of life.[1,2] Although AD is primarily driven by type 2 inflammation, whereas HS is characterized by type 1 and type 3 inflammatory responses, recent findings suggest the presence of several shared pathogenic mechanisms between these two conditions.[1,2] In detail, a skin barrier defect has been observed in both diseases, which is caused by qualitative and quantitative alterations of the lipids of the stratum corneum as well as dysregulation of antimicrobial peptides and alterations in the activity of the enzyme y-secretase.[1-4] Several lines of evidence suggest that these skin barrier alterations may be both a consequence and a contributor to the development of type 2 inflammation. A restoration of the normal physiological mechanisms of the skin barrier under treatment with dupilumab, a fully humanized monoclonal antibody that inhibits the IL-4 and IL-13 signaling, has been widely demonstrated in patients with AD.[4] We report the case of a young adult man with concomitant HS and AD, who experienced a marked improvement of both conditions after treatment with dupilumab, thus suggesting a relevant pathogenic role of type 2 inflammation and skin barrier dysfunction in both AD and HS.

Case Report

A 29-year-old male with a two-year history of HS, concurrently diagnosed with moderate to severe AD, was examined for exacerbation of both diseases. The patient had been previously treated with topical and systemic antibiotics, topical and systemic corticosteroids, and cyclosporine, with no clinical benefit. At our observation, physical examination showed the presence of a follicular phenotype of HS with papules, nodules, and scarring, located on the groin and axillae (Figure 1a). In addition, eczematous lesions consistent with AD were observed on the trunk and neck (Figure 2a). Eczema Area and Severity Index (EASI) score was 24.6, whereas HS severity was moderate

(Hurley stage II). Skin manifestations were associated with intense itching and severe pain (itch-Numeric Rating Scale [NRS]: 8; pain-NRS: 7). The patient's health was otherwise good, and blood examination (complete blood count, transaminases, blood glucose, creatinine, cholesterol, triglycerides, erythrocyte sedimentation rate, and C-reactive protein) revealed no abnormalities, with the exception of an elevated total IgE level (2567 kIU/L; normal values: 0-100). Dupilumab was administered at the induction dose of 600 mg subcutaneously, followed by a maintenance dose of 300 mg every 2 weeks, in association with an 8-week course of oral clindamycin. After 8 weeks of treatment, an almost complete regression of AD lesions was observed (EASI 2; itch-NRS: 2; pain-NRS: 1) and a concurrent marked improvement of HS lesions on the groin and axillae was also observed. After 16 weeks of dupilumab treatment, despite the discontinuation of antibiotic therapy, HS lesions were almost completely resolved (Figure 1b). Furthermore, a complete remission of AD (EASI 0; itch-NRS: 0; pain-NRS: 0) was observed (Figure 2b), associated with decreased IgE levels (452 kIU/L). The optimal control of HS manifestations achieved with dupilumab was maintained during 54 weeks of treatment, whereas the patient experienced a few AD relapses, which were successfully managed with topical steroids.

Discussion

HS and AD are two chronic inflammatory skin diseases that epidemiologically display a mutual association.[1,2] Indeed, patients diagnosed with HS have a 2-fold higher risk of developing AD compared with the control group (hazard ratio [HR]: 2.06; 95% confidence interval [CI]: 1.64-2.58). Conversely, a 40% increase in the odds of developing HS was reported in patients with a history of AD.[2]

This close epidemiological correlation suggests the existence of potential pathophysiological links between the two disorders, which may involve dysregulation of Notch signaling, epidermal barrier defects, and antimicrobial peptide dysfunction.[1,2] As recently suggested, a pathogenic link underlying both immune disorders may be caused by alterations in sphingolipid metabolism that are detected in both diseases.[3] Reduced levels of sphingomyelin and ceramides, with concomitant disruption of the skin barrier, were observed in both diseases, thus provoking the activation of the inflammatory cascade and the consequent release of tumor necrosis factor-α and other inflammatory mediators.[3]

In this regard, the restoration of the physiological lipid content of the skin barriers by dupilumab may explain the therapeutic success observed in our patient and in a few other cases of HS reported in the literature, as they all, to date, report a marked improvement in skin lesions and a reduction of flares during treatment.[5-7]

The case of our young male patient affected by concomitant HS and AD, who was successfully treated with dupilumab initially associated with systemic clindamycin, is similar to a recent report describing a 25-year-old man with severe AD and HS (Hurley stage III) who obtained good control of both diseases during a 1-year treatment with dupilumab, combined for the first 16 weeks with systemic clindamycin.[5] In our patient, the marked improvement in HS lesions during the initial phase of treatment with dupilumab may be partly due to the concomitant use of systemic clindamycin during the first 8 weeks. However, the patient's previous unsuccessful use of several systemic antibiotics, including clindamycin, and the maintenance of long-term remission of HS signs and symptoms for up to 54 weeks after clindamycin discontinuation suggest that dupilumab may have played an important role in both the induction and maintenance of the clinical response.
Notably, in two other patients, a substantial benefit in both HS and AD lesions was reported with the use of dupilumab as monotherapy after 4 and 6 weeks, respectively.[6,7]
Randomized clinical trials including patients with isolated HS, without coexisting AD, would be essential to clarify the therapeutic potential of dupilumab in this condition.

Conclusions

In conclusion, our case report provides further evidence of the existence of common pathogenetic mechanisms between HS and AD, suggesting a potential pathogenetic role of IL-4 and IL-13 in the pathogenesis of HS.

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Figure 1. Papular and nodular inflammatory lesions on the left thigh at baseline (**a**); marked improvement of HS lesions after 16 weeks of dupilumab therapy (**b**).

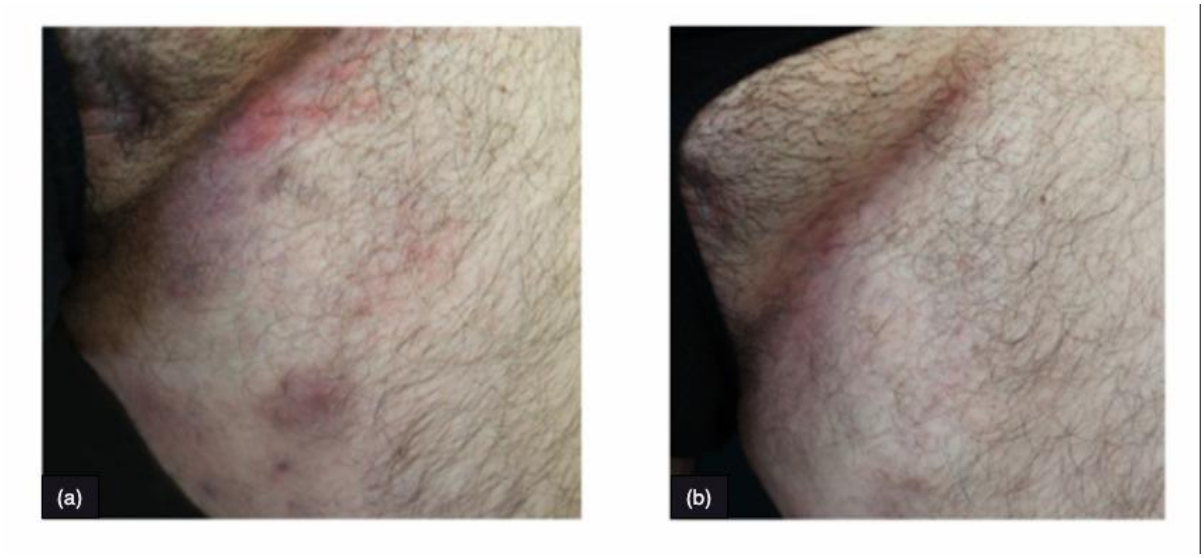

Figure 2. Widespread eczematous lesions located on the back at baseline (**a**); almost complete resolution of eczematous lesions after 16 weeks of dupilumab therapy (**b**).