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dupilumab and switched to 30 mg of upadacitinib. After 4 months of therapy, a complete clinical response was obtained and she noted a dramatic patchy hair regrowth of the vertex (Figs. 1G–H). Upadacitinib was well tolerated by our patients, without adverse effects. The central role of cytokines and Janus kinase (JAK)/signal transducer and activator of transcription (STAT) pathways in the pathogenesis of atopic dermatitis allowed the development new therapeutic options.² JAK1 plays an important role in the expression of interleukin 4 (IL-4), IL-5, IL-13, and IL-31. All of these cytokines are associated not only with AD but also with AA.³ This knowledge has brought in the theoretical use in a therapy with dupilumab in AA. The relationship of dupilumab and AA is controversial⁴; on the contrary, JAK inhibitors seem to have a role in all diseases in which the JAK/STAT pathway is involved. Several data on the literature and clinical trials have shown promising results of using JAK1 inhibitors for the treatment of AD and theoretically on AA as they share pathogenetic aspects.⁵ In agreement with these data, our experience suggests that upadacitinib may be a therapeutic option not only for AD but also for AA, which have failed conventional therapy. Investigations into JAK inhibitors are required to determine the long-term safety efficacy in AD and to elucidate the pathogenesis of AA.

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Concurrent Atopic Dermatitis and Psoriasis Successfully Treated With Brodalumab

To the Editor:

Atopic dermatitis (AD) and psoriasis (PsO) are the 2 most common chronic inflammatory skin diseases.¹ Th17 (T helper 17) and Th22 cells play a significant role in AD pathogenesis. Therefore, interleukin 17 (IL-17) may become the target for future biological therapies in AD.² To our knowledge, we report the first case of PsO and AD resistant to conventional therapies that was successfully treated with brodalumab, an IL-17RA (IL-17A receptor) inhibitor. A 68-year-old man came to our department with a medical history of a liver transplant performed in 2002 for hepatitis C virus–related cirrhosis, which is currently being treated with tacrolimus. Dermatological examination was significant for erythematous-squamous plaques on his bilateral lower limbs, lumbar area, elbows, and trunk, leading to the diagnosis of PsO (Fig. 1A). Moreover, numerous itchy, eczematous lesions were present on the flexural surfaces, scalp, face, and neck compatible with the diagnosis of AD (Fig. 1B). We carried out patch testing with negative results. We performed a skin biopsy in different body areas. Some of them were performed on the lumbar area, lower limbs, and trunk and showed psoriasiform hyperplasia of the epidermis, hypogranulosis, papillomatosis, and parakeratosis. These histopathologic features confirmed the diagnosis of plaque PsO (Fig. 1C). Other biopsies were carried out on the left antecubital area and on the scalp, and histological examination showed a chronic superficial dermatitis characterized by acanthosis, marked spongiosis, and exocytosis. The above morphological features were consistent with a diagnosis of AD (Fig. 1D). These clinical and histopathological features confirmed the diagnosis of severe PsO and the synchronous presence of a moderate form of AD. We chose to start a UV-B narrowband phototherapy, together with topical corticosteroid. After 3 months of treatment, there was no improvement in both clinical conditions. For this reason, we switched to 20 mg of methotrexate per week for 4 months

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The patients in this article have given written informed consent to the publication of their case details.

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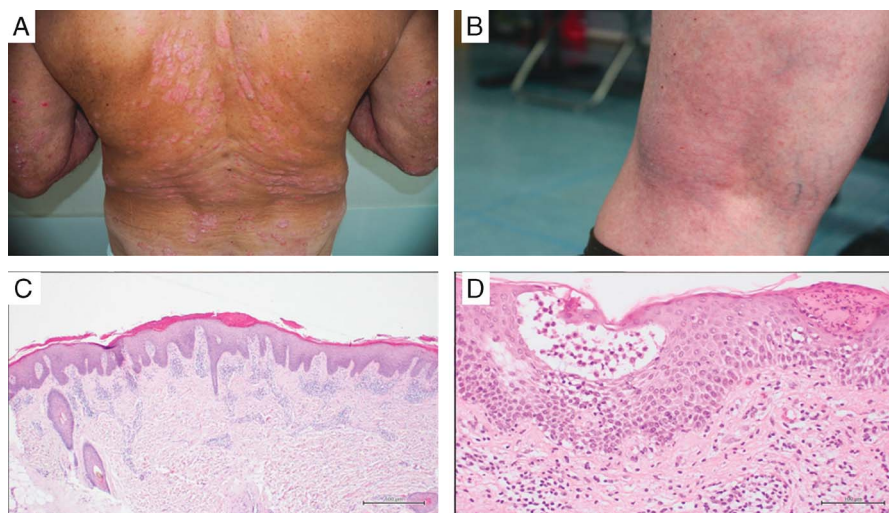


Figure 1. A, Severe PsO, with Psoriasis Area Severity Index score of 16 and body surface area score of 12. B, Moderate form of AD involving the flexural surface. C, Psoriasis: scanning view shows psoriasiform hyperplasia of epidermis—acanthosis and papillomatosis with saw-tooth appearance and clubbing of rete pegs (hematoxylin-eosin stain, original magnification $\times 5$). D, Eczematous dermatitis: the epidermis looks irregularly and mildly hyperplastic, with acanthosis and marked spongiosis associated with lymphocytic exocytosis and formation of intraepidermal vesicles (hematoxylin-eosin stain, original magnification $\times 20$).

with little improvement. We suggested using cyclosporine instead of tacrolimus as an antirejection therapy, but the hepatologist advised against this to avoid any adverse effect. Taking this into account, after informed consent, we started treatment with 210 mg of brodalumab by subcutaneous injection at weeks 0, 1, and 2, followed by 210 mg every 2 weeks. A complete clinical response was obtained after 8 weeks of brodalumab. Different studies support the theory of involvement of T_H17 cells and IL-17 in the pathogenesis of several chronic inflammatory skin diseases.³ However, in AD, the role of T_H17 cells has not been fully understood. T_H17 cells play a potential role in immune activation and are involved in the inhibition of filaggrin synthesis by keratinocytes. This causes disorders of keratinization and impaired skin barrier function. The evidence suggests that, in both AD and PsO, IL-17C overproduction may cause inflammation and psoriasiform hyperplasia.⁴ Brodalumab is the only anti-IL-17 treatment that affects not only IL-17A and IL-17F, but also IL-17C.⁵ Consequently, brodalumab could have beneficial effects not only for PsO but also for AD. In our case, the IL-17RA inhibitor brodalumab was effective in the treatment of AD. Further studies are required to evaluate the efficacy and safety of IL-17C antagonists in AD.

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(artt. 19 e 47 del D.P.R. 445/2000)

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in via Eleuterio n° 40

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Data, 14-06-2022

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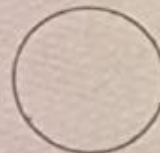


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