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Baricitinib improved morphoea manifestations in a patient with alopecia areata universalis

Dear Editor, Morphoea is a chronic inflammatory and fibrosing disorder affecting the skin and the subcutaneous tissue, characterized by an increased deposition of collagen and extracellular matrix. The onset symptoms include rounded erythematous–oedematous plaque with a lilac ring that gradually disappears and evolves into sclerosis; therefore, the skin has a pearly white look. Disease activity can be identified through physical examination and with the Localized Scleroderma Assessment Tool (LoSCAT), a scoring method that evaluates skin thickness, inflammation, lesion extent and damage.[1] Morphoea can have a significant impact on quality of life, influencing the individual's overall wellbeing by causing burning, pain and itching. The level of disease activity, the depth of involvement and the area of body surface affected determine the most appropriate treatment approach.

Here we report a case of morphoea in plaques that demonstrated a good response to baricitinib 4 mg daily. In December 2023, a 68-year-old man presented with generalized morphoea in plaques that developed in 2022 and were treated initially with topical clobetasol associated with methotrexate, and successively with mycophenolate mofetil and methylprednisolone, which were then suspended due to lack of clinical benefit. The patient had also been affected by alopecia areata universalis (AA) [Severity of Alopecia Tool (SALT) 100] for over 10 years, which had been unresponsive to systemic corticosteroids and other immunosuppressants.

On physical examination, he presented with roundish erythematous–oedematous plaques with characteristic a lilac rings that were nonliftable in fold, located on the trunk and inguinal region, and characterized by pruritus with a numerical rating scale for pruritus (NRSp) score of 6 and a LoSCAT score of 12 (Figure 1). Due to the lack of efficacy of previous treatments and the association with AA, oral baricitinib 4 mg daily was prescribed in combination with topical clobetasol ointment applied once daily for 20 days, followed by twice-weekly application on nonconsecutive days. After the first month of baricitinib, the patient reported the prompt disappearance of itchiness (NRSp 0), and a reduction in the infiltration and erythema of the lesions (LoSCAT 8). After 4 months of treatment, a significant improvement in morphoea plaques was documented (LoSCAT 5). At the 7-month' follow-up, further improvement in the morphea plaques was documented (LoSCAT 2) (Figures 2, 3). There was also documented partial regrowth of the eyebrows and beard, while no substantial improvement was noted in AA (SALT 96). The patient did not report any side effects.

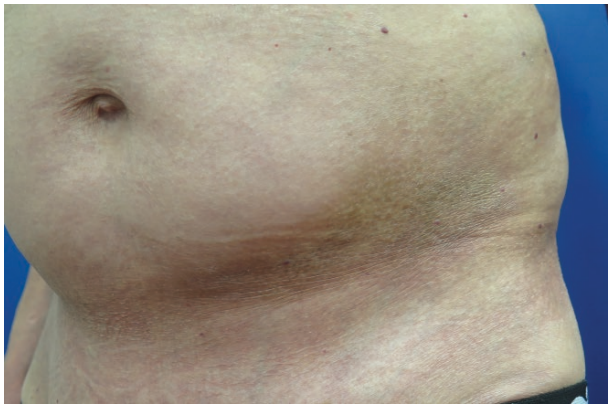

Figure 1 Efficacy of baricitinib in a patient with morphoea. Photograph taken during the first clinical visit: erythematous infiltrative plaque on the left abdominal region Localized Scleroderma Assessment Tool score 12.

The treatment of limited morphoea plaques is based on topical high-potency corticosteroids, topical tacrolimus, topical vitamin D analogues and intralesional corticosteroid injections. Systemic therapy is often justified for patients with extensive, rapidly progressing active morphoea and is based on the use of systemic steroids, associated with methotrexate 15–25 mg weekly or mycophenolate mofetil 1–2 g daily if the former is not effective or not tolerated. In our patient, after the failure of previous treatment, we decided to initiate therapy

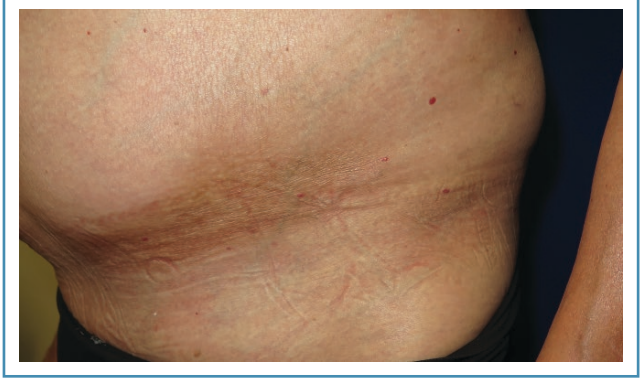

Figure 2 Photograph taken during the seventh month of treatment with baricitinib 4 mg daily, with an important reduction in the erythematous-infiltrative component of the plaque Localized Scleroderma Assessment Tool score 2.

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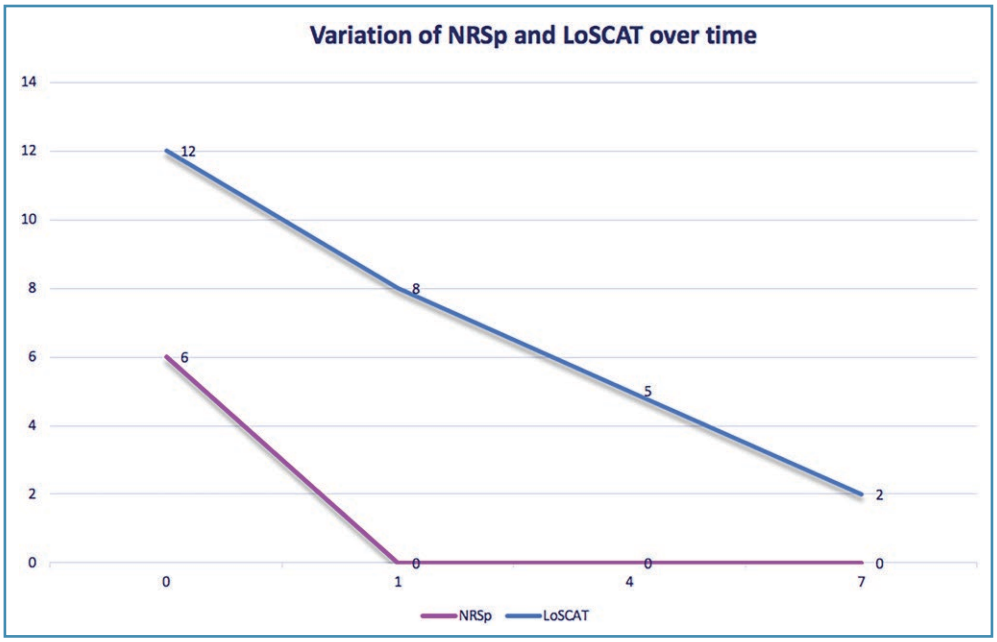

Figure 3 Reduction in numerical rating scale for pruritus (NRSp) and Localized Scleroderma Assessment Tool (LoSCAT) scores during treatment with baricitinib 4 mg daily.

with baricitinib, a selective and reversible Janus kinase (JAK) inhibitor, approved for the treatment of rheumatoid arthritis, atopic dermatitis and AA, for the coexisting morphoea and AA. Two case reports highlight the efficiency of baricitinib in the treatment of morphoea.[2,3] Moreover, a literature review, analysing 11 cases of morphoea and 15 cases of systemic scleroderma treated with JAK inhibitors, demonstrated their efficiency in reducing the infiltration of morphoea plaques, as well as cutaneous fibrosis in scleroderma. The association between alopecia areata and morphoea, both inflammatory disorders with a T-cell-mediated autoimmune pathogenesis, is not extensively described in the literature and, as in our case, may be coincidental.[4] In our case, the duration of AA may explain, at least in part, the lack of response to baricitinib. Our therapeutic focus was primarily directed toward a potential response of morphoea to baricitinib.

The therapeutic rationale arises from the analysis of the role of JAKs, which have been shown to be downstream of profibrotic signalling mediated by transforming growth factor (TGF)-β.[5] The use of JAK inhibitors has demonstrated the inhibition of the effects mediated by TGF-β in cutaneous sclerosis, *in vitro* and *in vivo*.[6] Therefore, the JAK/signal transducer and activator of transcription pathway seems to play an important role in the pathogenesis of morphoea; accordingly, clinical and experimental data suggest the potential efficiency of JAK inhibitors in morphoea and systemic scleroderma. For these reasons, baricitinib could represent a valid therapeutic option for morphoea; however, further clinical studies are needed to increase the number of cases and confirm the actual benefit.

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