Full histological and clinical regression of morphea with tofacitinib

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Morphea is a fibrosing skin disease with varied forms of clinical presentation. The most common is the presence of one or more localized indurated plaques. Treatment of morphea can be challenging. Prednisone and methotrexate are the mainstay of treatment but its efficacy is quite variable. We recently treated a 59-year-old male with localized morphea in one of the legs that failed to remit after extensive periods of therapy initially with prednisone 40 mg daily for 2 months and then the combination with 15 mg weekly methotrexate for an additional 2 months. We treated our patient with tofacitinib a JAK 1,3 inhibitor and discontinued steroid and methotrexate. As demonstrated on Figs. 1 and 2, the marked induration after 3 months of continuous treatment with 10 mg of tofacitinib daily showed histological and complete clinical regression, and it is maintained until the present moment. Ruxolitinib a JAK 1, 2 inhibitor has shown to induce remission in sclerodermatous cutaneous disease. Overexpression of JAKs was recently described in sclerodermatous tissues and improved disease in mice with tofacitinib [1–3]. The observations here reported suggest that JAK inhibition maybe a promising form of treatment for fibrous skin disease. Beneficial effect of tofacitinib in other less common skin autoimmune diseases has been recently demonstrated by our group [4, 5].

Patient consent was obtained.

Fig. 1 **a** Clinical photograph showing the indurated erythematous plaque of morphea before and **b** after treatment with tofacitinib

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Fig. 2 Microphotograph of skin biopsy showing extensive sclerosis in dermis that responded to treatment with tofacitinib. Before (**a**, H and E staining, $\times 40$) and after (**b**, H and E stain, $\times 100$) treatment

Compliance with ethical standards

Disclosures None.

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