Letter

Baricitinib for Refractory Eosinophilic Fasciitis: Myth or Reality?

To the Editor:

We read the recent article by Sehgal et al with great interest.¹ The authors described a 37-year-old patient with eosinophilic fasciitis (EF) who showed no improvement after an initial aggressive treatment strategy (intravenous [IV] methylprednisolone, prednisone, methotrexate, mycophenolate mofetil, and IVIG). Subsequently, the use of the Janus kinase (JAK) 1/2 inhibitor baricitinib resulted in considerable improvement in the patient's symptoms and skin appearance. However, there are some details that need further clarification.

First, it is not clear whether imaging, such as magnetic resonance imaging (MRI) of the extremities, was performed before the diagnosis of EF. This is often performed before patients proceed to deep biopsy. Second, the authors used the Health Assessment Questionnaire II score as well as ultrasound assessment of fascial thickness and echogenicity of the gastrocnemius to illustrate the efficacy of baricitinib. The modified Rodnan skin score (mRSS), a validated and reliable measure of skin involvement in systemic sclerosis, is frequently used in clinical practice for EF.² Therefore, other assessment methods such as the mRSS, MRI of the extremities, joint mobility, and the percentage of decreased peripheral blood eosinophils may be more helpful in assessing efficacy. Third, the dose of baricitinib usually consists of 4 mg per tablet and 2 mg per tablet.³ Information about the dose of baricitinib used, the longer-term plan for use of baricitinib, the intended treatment duration, and the presence of adverse drug reactions (such as major adverse cardiovascular events, malignancy, and thrombosis) should be described in detail, as these will be instructive and informative for subsequent treatment in the same situation.⁴ Finally, we add the mechanism of baricitinib to explain its effects. Baricitinib is a novel oral small-molecule JAK inhibitor that targets the intracellular JAK/signal transducers and activators of transcription (STAT) signaling pathway and achieves antiinflammatory and immunomodulatory effects by inhibiting JAK phosphorylation, thereby blocking STAT phosphorylation; this results in reduced synthesis of downstream

inflammatory cytokines, which in turn inhibits CD4⁺ T cell proliferation and blocks the synthesis and secretion of a variety of inflammatory cytokines.⁵

In summary, although some of the details in this article need to be further elucidated, this study reveals a successful case of baricitinib in the treatment of EF and supports growing interest in the use of baricitinib in sclerosing skin disorders.

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The authors declare no conflicts of interest relevant to this article.

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