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CASE REPORT

Tofacitinib for Prurigo Nodularis: A Case Report

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Purpose: Despite recent advances in the treatment of prurigo nodularis, conventional treatment suffers from a dilemma of poor efficacy. The clinical use of Janus kinase (JAK) inhibitors in the treatment of Prurigo nodularis has rarely been explored.

Patients and Methods: We present a case of prurigo nodularis successfully treated with, JAK inhibitor tofacitinib with no adverse effects.

Results: This case report of successful treatment shows a good clinical efficacy of using JAK inhibitor tofacitinib in the treatment of prurigo nodularis. Cytokines may be an important cause of prurigo nodularis.

Conclusion: JAK inhibitor tofacitinib may be a new option for the treatment of prurigo nodularis, especially for patients who have failed conventional treatment.

Keywords: prurigo nodularis, JAK inhibitors, tofacitinib

Introduction

Prurigo nodularis (PN) is an intensely pruritic disease characterized by highly keratinized, itchy papules and nodules symmetrically distributed on the extremities of the limbs.[1] Treatment is often challenging and unsatisfying. Recently, Janus kinase (JAK) inhibitors has been successfully used for the treatment of PN.[2,3] Here, we report a case of a patient with PN who did not respond to conventional treatment but was successfully treated by the JAK inhibitor tofacitinib.

Case Presentation

A 65-year-old male patient was admitted due to " recurrent systemic erythema, papules and itching for more than 2 years" (Figure 1A1–D1). His laboratory examination shows no obviously abnormal (Including humoral immunity, autoimmune antibody profile, tuberculosis, hepatitis B, AIDS, syphilis, etc.) except the eosinophils were $1.47*10^9$/L, and the percentage of eosinophils was 22.1%. Over the past two years, he has undergone many diagnoses and treatments without satisfactory results. Eight months before this admission, the patient received a skin pathological examination in another hospital in the city and was diagnosed as PN. After admission, the patient was diagnosed as PN, and pruritus Numerical Rating Scale (NRS) is 10 (Figure 1E1), Dermatology Life Quality Index (DLQI) is 23 (Figure 1E1). We referred to the guidelines for the treatment of PN and treated the patient with the following:[4] topical corticosteroids (topical halogen cream), NB-UVB irradiation, and systemic therapy (glycyrrhizic acid, vitamin C, calcium gluconate, rubastine fumarate, ebastine, pregabalin, and thalidomide). However, the symptoms did not improve significantly. Report on the effectiveness of dupilumab in PN,[4–6] we had planned to give him dupilumab, but he refused because of the expensive price. Then, we treated the patient with tofacitinib and stopped other treatments. After 1 week of tofacitinib application, the number of nodules decreased and became smaller, and the NRS and DLQI also improved significantly. Refer to the dosage of tofacitinib in the treatment of pruritic dermatosis,[2,7,8] the patient took tofacitinib for a total of 6 weeks at a dosage of 5mg bid for weeks 1–2, 5mg qd for

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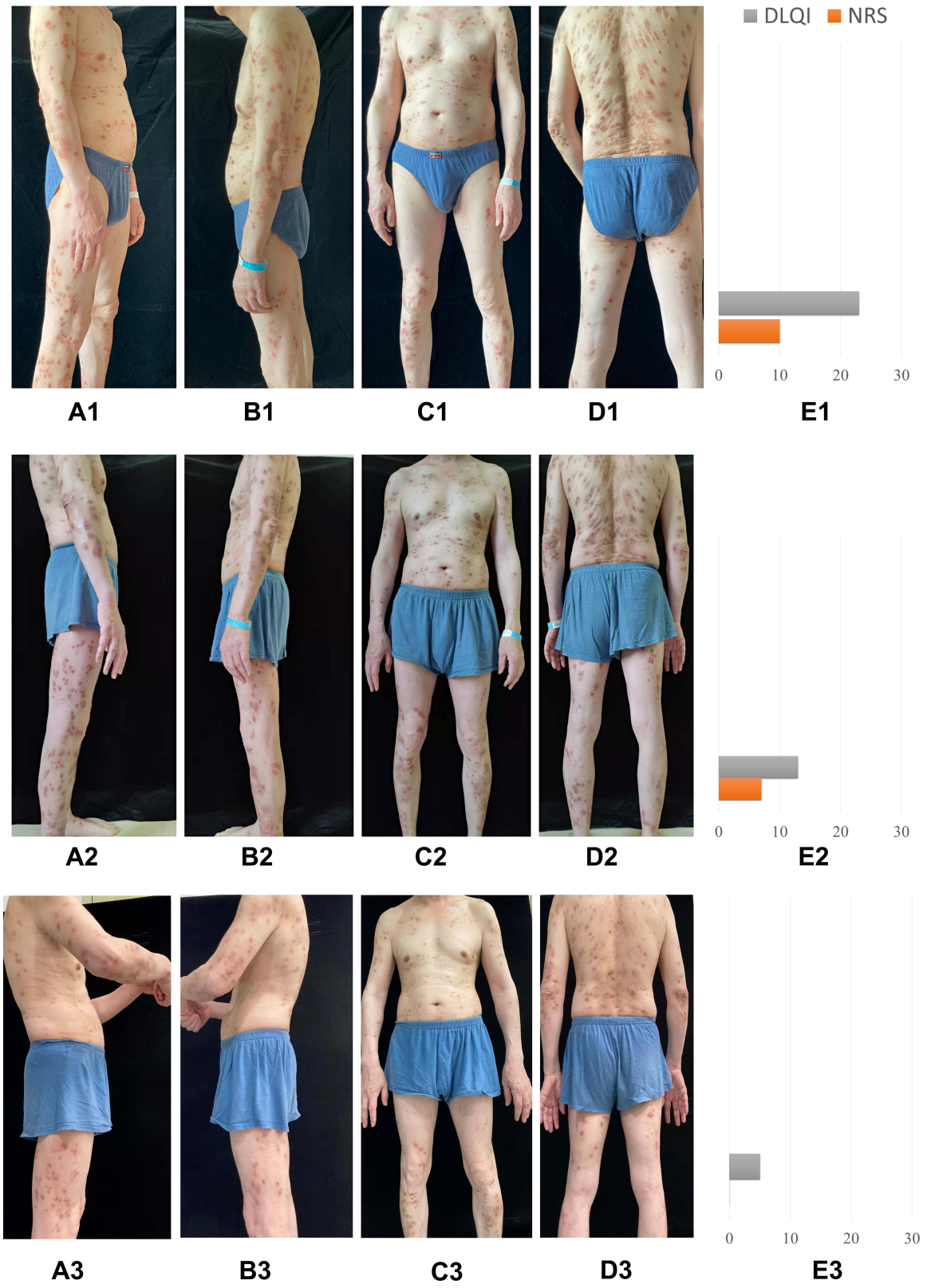

Figure 1 Changes of skin lesions and NRS, DLQI during tofacitinib treatment. (**A1 ~ E1**) Images of skin lesions and NRS, DLQI when patient started oral tofacitinib. (**A2 ~ E2**) Images of skin lesions and NRS, DLQI at week 1. (**A3 ~ E3**) Images of skin lesions and NRS, DLQI at week 6.

weeks 3–4, and 5mg qod for weeks 5–6. Skin lesions were photographed before, 1 week after, and 6 weeks after tofacitinib treatment (Figure 1A2–D2; Figure 1A3–D3), NRS and DLQI were assessed simultaneously (Figure 1E2; Figure 1E3). In view of previous reports of exacerbations of PN treated with tofacitinib,[8] we closely observed the patients and showed good efficacy with no adverse effects during 6 weeks of tofacitinib administration. The patient was followed up for 5 months after drug withdrawal, and no recurrence was found.

Discussion

PN is a refractory skin disease, and pruritus is the main problem that needs to be solved. According to recent studies, the pathogenesis of PN is associated with decreased intraepidermal nerve fiber density and abnormal secretion of cytokines such as IL-4, IL-17, IL-22, and IL-31.[1,2] As a non-selective JAK inhibitor, tofacitinib can inhibit the JAK-STAT pathway, block the transcription of IL-4 and IL-31,[9,10] increase the density of epidermal nerve fibers, and reduce pruritus. The first reported case of tofacitinib for PN,[2] the success of baricitinib for prurigo-type atopic dermatitis,[3] both demonstrate the effectiveness of the JAK inhibitor for the treatment of PN.

Conclusion

Therefore, tofacitinib as an inhibitor of JAK is a promising choice for the treatment of PN, in which cytokines are the main inflammatory factors causing pruritus. In future, larger sample sizes and multi-ethnic clinical studies will be needed to provide stronger evidence.

Ethics Statement

Signed consent was obtained from the family member for the publication of the case details and accompanying images. Institutional approval was not required to publish the case details.

Consent Statement

Written informed consent was provided by the patient for publication of images and information.

Disclosure

Tianhao Li and Pingsheng Hao are co-correspondence authors for this study. The authors have no conflicts of interest to declare in this work.

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