Successful treatment of refractory palmoplantar psoriasis in a psoriatic arthritis patient with the JAK inhibitor tofacitinib

Sirs.

Palmoplantar psoriasis (PP) is an incapacitating condition that significantly impairs quality of life. PP prevalence is between 2–40% among psoriasis and psoriatic arthritis (PsA) patients (1). It tends to be refractory to conventional therapies and may last for several years causing significant impairment in mobility and agility. (2). One study reported that just 27.4% of PP patients showed improvement with topical agents, whereas the remaining patients required systemic therapies (3).

This report details a 55-year-old male Caucasian patient with refractory PP, diagnosed with psoriasis vulgaris at the age of 15, while PsA started at the age of 52. The initial treatment targeting psoriasis vulgaris was oral methotrexate from 2011 to 2016 with success. Suddenly he presented a persistent psoriasis flare, resistant to methotrexate. In month 4 of treatment, the patient suddenly developed intense erythema, scaling and deep fissuring of the central region of palms and soles, with generalized psoriatic lesions including ESR and CRP. Various factors including smoking, stress and trauma can trigger PP in genetically susceptible individuals leading to excessive cytokine production and keratinocyte proliferation. Inducible IL-23 expression can trigger a TH17 response with local production of TNF-α, IL-17, and IL-22 that induce keratinocytes stimulation and recruitment of neutrophils. Neutralisation of TNF-α, IL-23 and IL-17A has demonstrated to be effective in patients presenting with PP who do not respond to topical or other systemic medications (3-7). In our patient, targeting these cytokines one by one, turned out to be insufficient, and TNF inhibition even triggered PP lesions, as previously described (4).

Tofacitinib inhibits Janus kinases JAK1, JAK2 and JAK3, involved in the signalling of more than one cytokine present in psoriasis (IL-23, IL-22 and IFN-gamma), hence, the complete remission observed in this refractory PP patient indicates that downregulation of the signaling of more than one individual cytokine by tofacitinib prevents potential escape mechanisms of inflammation during single cytokine targeting. To the best of our knowledge, this is the first case report of a successful therapy of a patient with PP using tofacitinib.

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