Rapid Improvement of Palmoplantar Psoriasis after Cessation of Smoking



Sir,

Palmoplantar psoriasis is a condition characterised by erythema, infiltration, fissuring, and scaling, and is sometimes associated with recurrent crops of sterile pustules, occasionally presenting with hyperkeratosis. This chronic inflammatory skin disease can be disabling in cases where patients experience painful fissuring and may affect daily activities, possibly resulting in debilitating pain.¹

Smoking has been observed to be a trigger in the development of this form of psoriasis through oxidative, inflammatory, and genetic mechanisms. Nicotine damages skin by increasing the reactive oxygen species and decreasing the expression of antioxidants. It also stimulates innate immune cells integral to the pathogenesis of psoriasis including macrophages, keratinocytes, and dendritic cells.² A better understanding of palmoplantar psoriasis has disclosed some differences when compared to *psoriasis vulgaris* regarding age of onset, female predominance, and smoking influence.³ The impact of the cessation of smoking during the clinical course of the disease remains to be established. We present a case of refractory palmar psoriasis with clinical improvement after cessation of smoking.

A 51-year-old male farmer presented to the Department of Dermatology at San Cecilio University Hospital, Granada, Spain, with palmoplantar psoriasis. He had been suffering this condition since the age of 34 with periods of remission and aggravation. His medical history showed that he had been a heavy smoker (3 packs per day) for the last 35 years. His lesions had become extremely painful and incapacitated him to the extent that he was unable to work. An examination revealed scaly hyperkeratotic plaques with fissures on both hands [Figure 1]. His nails were also dramatically affected showing distal onycholysis, subungual hyperkeratosis, Beau's lines and oil spots [Figure 2]. His feet were free of lesions. Several therapies were used including topical steroids, psoralen and ultraviolet A (PUVA) light therapy, and methotrexate, but no improvement was observed.

Psoriasis on hands and feet is a disabling condition which highly impacts quality of life (QOL). It is a particularly challenging form of psoriasis due to its resistance to treatment; topical therapies usually fail because of the thickened horny layer of epidermis in these areas, and systemic conventional therapies do



Figure 1: Tender scaly plaques on palmar side of the hands. The patient was unable to work.



Figure 2: Nails were dramatically involved showing distal onycholysis, subungal hyperkeratosis, Beau's lines, and oil spot.

not always give a therapeutic gain. Many of these systemic therapies used in plaque psoriasis have been used in palmoplantar psoriasis, but none are generally accepted as being reliably effective. In addition, patients with this condition are excluded from most clinical trials of psoriasis, which leads to no evidence-based consensus regarding a therapeutic strategy for this disease. With all these considerations in mind, we also have to consider other environmental conditions that have an effect on the therapeutic results of smoking. Patients with psoriasis are more likely to develop hypertension, obesity, depression, or psychiatric disorders, and become drinkers or smokers due to the impact of this disease on their QOL.^{4,5}

Tobacco use is preventable, and has been shown to exacerbate pre-existing palmoplantar psoriasis, but its use has also been found among subjects with new onset of the disease.⁶ Moreover, smoking has been suggested as an aggravating factor in the treatment outcomes and is believed to drive disease severity.^{6,7} The pathogenic mechanism is uncertain but may be linked to the products of smoking which induce oxidative stress, encouraging inflammatory cells to accumulate in the epidermis, altering the function of these cells and the extracellular matrix turnover.⁸ Smoking enhances expression of genes known to confer an increased risk of psoriasis (HLA-Cw6, HLA-DQA1*0201 and CYP1A1) and initiates the formation of free radicals that stimulate cell-signalling pathways active in psoriasis, including mitogen-activated protein kinase, nuclear factor-kB, and Janus kinase/signal transducers and activators of transcription. Nicotine stimulates cells associated with the pathogenesis of psoriasis such as dentritic cells, macrophages, and keratinocytes. These cells release cytokines which activate T-lymphocytes and perpetuate a cycle of chronic inflammation.³

Improved understanding of the possible link between smoking and palmoplantar psoriasis may provide further insight into mechanisms underlying smoking, psoriasis, and its improvement after cessation of smoking. In a case series study, up to 95% of patients with psoriasis of the hands and feet were former or current smokers, but the impact of cessation in the clinical course of the disease has not been clarified yet.⁹ In our case, a satisfactory response to methotrexate (15 mg/week) and emollients was observed only after cessation of smoking. Physicians should keep this in mind and carefully investigate the possibility of a patient being a smoker if that patient is refractory to conventional therapies. In unresponsive cases of psoriasis, smoking may be responsible for the refractoriness. Discontinuation of smoking may lead to a satisfactory response, as was seen in our patient.

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