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Letter to the Editor

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Plaque Psoriasis Formation and Body Hair (Re)Growth: A Common Pathway Activation?

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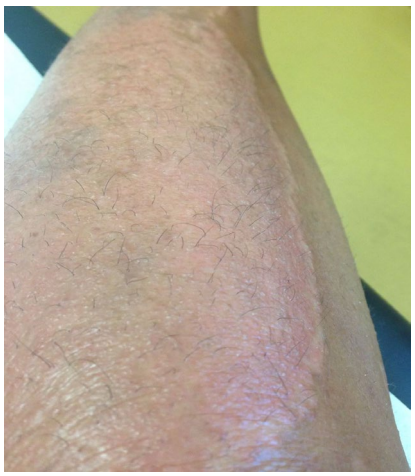
To the Editor,

We report the case of a 39-year-old woman suffering from severe psoriasis over a long-term. In the past the patient had been treated with cyclosporin, methotrexate, adalimumab and with poor results and had reached a good control of the disease only upon treatment using ustekinumab. After 5 years of clinical remission biological therapy with ustekinumab was discontinued because the patient was planning a pregnancy. Shortly after, a sudden worsening of the skin condition was observed, with the development of new psoriatic plaques, especially on the lower limbs. In this occasion the patient noticed that body hair density was greatly increased in the new psoriatic plaques of the lower limbs (Figure 1). Due to the severity of psoriasis recurrence, after few weeks the patient decided to renounce pregnancy and therapy with ustekinumab was resumed. Psoriasis plaques resolution was observed in few weeks, and the hair of the lower limbs started to grow back homogeneously. The effect of psoriasis on hair growth is not known, and there have been reports of both loss of hair and increased hair density associated with psoriatic plaques. In 1972 Shuster et al[1] first described hair loss affecting both the scalp and the psoriatic plaques of the body, and coined the definition of psoriatic alopecia. According to their paper psoriatic alopecia could manifest itself as a generalized telogen effluvium, as scarring alopecia or, more commonly, as a decrease in hair density and thickness and an increased number of distrophic bulbs in psoriatic plaques. These findings were confirmed by other studies, which found a higher proportion of telogen and catagen hairs[2] and a lower percentage of anagen hairs[3] in lesional skin compared with healthy controls. Loss of visible hair in non-scalp psoriatic lesions was also observed by Rittiè et al,[4] who recently suggested that a key role in this phenomenon could be due to a down-regulated expression of a cytokine-

Figure 1: Hair Body in a Psoriatic plaque of the Lower Limb.



regulated set of sebaceous gland signature genes resulting in abnormal function and atrophy of sebaceous gland. On the other hand Sawan et al described a higher hair count in psoriatic areas of the scalp of 7 patients.[5] The hypothesis that both epidermal lesion of psoriasis and proliferation of hair matrix keratinocytes during the anagen phase may share some activating mechanisms was first proposed by Paus et al. in 1988,[6] who observed remarkable parallels between the Koebner phenomenon in psoriasis and wounding-induced anagen hair growth. Supporting this hypothesis, a study by Hampton et al[7] showed that the suprabasal psoriatic epidermis had an increased level of nuclear β-catenin compared with uninvolved or normal skin. β-Catenin is a protein involved in the control of the hair cycle, and its expression is correlated with the induction of the anagen phase and the differentiation of stem cells. Transiently activating β-catenin signaling in adult mouse epidermis has been shown to induce growth of new hair follicles.[8] Based on these findings, Sawan et al[5] suggested that scalp psoriasis may result in an increased recruitment of stem cells and a switch-on entry in the anagen phase. However this theory is controversial, since, according to a recent research,[9] some of the pro-inflammatory cytokines mainly involved in psoriasis pathogenesis such as interleukin-1, tumor necrosis factor-α, interferon-γ, and transforming growth factor-α are able to markedly induce Wnt5a expression. Overexpressed Wnt5a has been shown to suppress the expression and translocation of β-catenin during hair follicle regeneration. According to these data Wnt5a could suppress hair follicle regeneration by suppressing the activation of the canonical Wnt signaling pathway.[10,11] In conclusion, the relationship between Wnt5a and β-catenin signaling in psoriasis and hair growth is still unclear. Current evidence suggests that a complex interplay between Wnt5a and β-catenin could regulate the pathogenesis of both these conditions.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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