INTRODUCTION

We describe the case of a young woman who developed localized hypertrichosis and psoriasis on her left lower extremity following a fracture and plaster application. The sudden appearance of postcast hypertrichosis and post-traumatic psoriasis are well-known events if considered separately, but their concurrence is exceptional and may be explained by the expression of several growth factors and cytokines involved in the pathogenesis of both conditions.

CASE REPORT

A 23-year-old woman was seen because of excessive hair growth on a psoriasis plaque of her leg, following prolonged immobilization in a plaster. In fact, approximately 1 month before the visit, she was involved in a motor vehicle accident and fractured her leg.

She had a 9-year history of mild psoriasis vulgaris, successfully controlled by the topical application of coal tar and steroids. However, she had been completely free from psoriasis lesions for the last year without any treatment.

On physical examination, a round erythematous scaly plaque measuring 4 cm × 4 cm with tufts of hair was present on the extensor aspect of the left leg, localized on where the plaster had rubbed [Figure 1]. Excessive hair growth was not evident in any other body region. The clinical findings were consistent with both localized hypertrichosis secondary to plaster application and psoriatic isomorphic response.

During the subsequent 2 weeks, a topical mid-potency corticosteroid cream was applied two times daily on the lesion, with a prompt resolution of the erythema and scaling. The hypertrichosis gradually lessened within 2 months, and no additional treatment was needed.

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DISCUSSION

Post-cast hypertrichosis is a common transient occurrence in orthopedic patients and generally resolves within a few months. To exclude other possible causes of acquired localized hypertrichosis (drugs, infections, postinflammatory sequellae, scleroderma, vascular conditions, and postsurgery), a detailed patient history must be collected.\(^{[1‑3]}\)

The psoriatic hair alterations reported in the literature comprise temporary hair loss, scarring alopecia, ultrastructural abnormalities, and also two cases of localized hypertrichosis. However, in these cases, localized hypertrichosis occurred on more than one pre-existent psoriatic lesion, probably as a result of sustained arterial hyperemia due to chronic inflammatory stimuli, even if the exact underlying mechanism is still unknown.\(^{[4]}\)

In our patient, the appearance of a single psoriatic plaque and overlying localized hypertrichosis were simultaneous, opening new scenarios on the relationship between psoriasis and hair.

Hair involvement in patients with psoriasis is one of the most common events over the course of the disease (50%–80% of cases). Hair loss due to scalp psoriasis is frequent, but alopecia is almost always noncicatricial, and complete regrowth occurs when inflammation burns out.\(^{[5,6]}\) In our case, psoriasis did not negatively interfere with the hair cycle, on the contrary, it seemed to stimulate hair growth. We hypothesize that the psoriatic Koebner phenomenon was at the basis of both hair growth and plaque appearance, probably through shared signaling pathways influencing the hair cycle and the psoriatic inflammatory process simultaneously. The expression and production of growth factors and cytokines involved in this mechanism should be further investigated.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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