A case of inverse psoriasis successfully treated with adalimumab

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Abstract

Inverse psoriasis is a rare form of psoriasis characterized by the involvement of skin fold areas rather than the more common psoriatic involvement of the extensor surfaces of the extremities, trunk, and scalp. In addition, it requires a modified therapeutic approach because it is often less responsive to standard treatment regimens. Current treatment recommendations for inverse psoriasis mainly consist of topical agents, including corticosteroids, calcipotriol, and immunomodulating agents, whereas systemic medications remain insufficiently studied. Although adalimumab, a TNF-α inhibitor, has been approved for the treatment of moderate to severe plaque psoriasis, some reports indicate that TNF-α inhibitors may sometimes trigger psoriatic lesions, including inverse psoriasis. However, we present a case of inverse psoriasis and psoriatic arthritis unresponsive to standard treatment that was successfully treated with adalimumab.

Introduction

Inverse (intertriginous) psoriasis is characterized by the involvement of skin fold areas such as the inguinal folds, gluteal cleft, axillae, and external genitalia, rather than the more common psoriatic involvement of the extensor surfaces of the extremities, trunk, and scalp (1). Some reports have indicated that tumor necrosis factor-α (TNF-α) inhibitors may induce psoriatic lesions, including inverse psoriasis (2). In contrast, we present a case of successful treatment of inverse psoriasis in a patient with psoriatic arthritis (PsA) with the TNF-α inhibitor adalimumab.

Case report

A 37-year-old Caucasian male with psoriasis and a history of dactyliitis came to our rheumatology practice for assessment of hand oligoarthritis. He had painful and swollen joints for the last 2 years and psoriatic lesions affecting the scalp that had been recurring for more than 10 years and were treated using topical corticosteroids. His psoriasis worsened 3 years ago when the lesions extended to the intergluteal area and the genitalia. While fungal infection was excluded, a skin biopsy revealed histological alterations consistent with psoriasis. Thus, topical pimecrolimus was initiated in addition to the topical corticosteroids previously prescribed. Despite transient improvement, co-therapy with pimecrolimus and corticosteroids failed to achieve long-term clinical relief after 18 months of regular use. At the time of examination at the rheumatology practice, the laboratory tests were unremarkable and the rheumatoid factor and anti-citrullinated protein antibodies were negative. The diagnosis of PsA was established according to the Classification of Psoriatic Arthritis (CASPAR) study group criteria. Non-steroidal anti-inflammatory drugs were prescribed. Six months later, the PsA had progressed to the small joints of both feet. He was therefore started on a disease modifying therapy with methotrexate (MTX) 15 mg qwk. After three weeks of MTX therapy, the patient complained of adverse events, including severe nausea with vomiting, headache, and insomnia. His arthritis was still active (Disease Activity Index (DAS28-ESR) 3.80) and his quality of life was adversely affected due to arthritis (Health Assessment Questionnaire: Patient-Reported Outcomes Measurement Information (HAQ-PROMIS) 1.25) and inverse psoriasis (Dermatology Life Quality Index (DLQI) 15). Considering the unbearable adverse effects, MTX was discontinued.

Because adalimumab, a fully humanized monoclonal antibody against TNF-α, is currently registered for treatment of PsA as well as moderate to severe plaque psoriasis (3), therapy with adalimumab was begun according to the national guidelines for the treatment of PsA. At the beginning of the treatment, erythematous macules, papules, and plaques were observed on the glans and the outer aspect of the foreskin, and erythema with erosions in the gluteal fold (Fig. 1, Day 0). After 90 days of treatment with adalimumab 40 mg once every 14 days by subcutaneous injection, not only was the arthritis in remission (DAS28-ESR 1.39), but an almost complete regression of the psoriatic lesions was also observed. Only small residual erythematous macules and papules remained on the glans and mild erythema in the gluteal cleft (Fig 2, Day 90). The patient’s quality of life improved significantly (HAQ-PROMIS 0 and DLQI 1). After two visits to our rheumatology department during a 6-month follow-up, he is doing well, reports no adverse events of the adalimumab therapy, and has no psoriatic skin lesions.

Discussion

To our knowledge, this is the first report of successful treatment of inverse psoriasis with adalimumab. Indeed, our patient that received adalimumab for treatment of PsA concomitantly experienced almost a complete regression of intertriginous psoriasis, which had been resistant to conventional topical anti-psoriatic agents.

Inverse psoriasis presents in only 3 to 7% of psoriatic patients and differs from the more common psoriasis vulgaris in the localization of skin lesions as well as in the therapeutic approach. Namely, inverse psoriasis is less responsive to conventional therapy and often presents a therapeutic challenge (1). Due to the lack of internationally accepted guidelines for the treatment of inverse

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Psoriasis, and also a lack of our own national guidelines, current treatment recommendations are based on the recently published consensus that includes mainly topical agents, such as corticosteroids for acute exacerbations and calcipotriene (calcipotriol) or immunomodulating agents (pimecrolimus or tacrolimus) for long-term therapy. A combination of these approaches is sometimes needed (4). The potential role of microorganisms in producing an inflammatory reaction in inverse psoriasis should also be considered and some patients may need appropriate antimicrobial therapy (4). The benefits of systemic treatment options have not been sufficiently studied. Nevertheless, as advocated by some experts, the use of systemic medications, including biologics, may have a role in the management of inverse psoriasis when topical medications are ineffective (4). Efalizumab, a biologic formerly available to treat psoriasis, has been used in some patients with severe inverse psoriasis with good results (1, 5).

Adalimumab, a TNF-α inhibitor, has been approved for the treatment of PsA and also, more recently, for moderate to severe plaque psoriasis (3). The use of these medications has expanded the pharmacological options for the treatment of psoriasis and revolutionized the therapeutic landscape. On the other hand, TNF-α inhibitors might sometimes worsen preexisting psoriasis or even trigger a new-onset of psoriasis through unknown mechanisms (6). Our case indicates that adalimumab may represent a viable treatment option for patients with inverse psoriasis unresponsive to standard treatments.

**Conclusion**

We describe the first case of the successful treatment of inverse psoriasis with adalimumab. Furthermore, our observations suggest that adalimumab may be considered as a treatment approach in inverse psoriasis that is resistant to conventional therapy. However, further clinical studies are needed to support this observation.
References