Volume 21 Number 11
November 2015

Case presentation

Adalimumab-induced psoriatic alopecia/alopecia areata-like reaction in a patient with Crohn's disease

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Dermatology Online Journal 21 (11): 6

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Abstract

Anti-tumor necrosis factor (anti-TNF) agents have been successfully used to treat both chronic idiopathic inflammatory bowel disease and other immune-mediated chronic diseases, but they can also induce a wide array of cutaneous reactions, including new-onset psoriasis and alopecia. We report a case of alopecia associated with psoriasiform skin lesions in a patient on adalimumab treatment for Crohn's disease.

Keywords: Adalimumab; Anti-tumor necrosis factor-alpha; Alopecia; Alopecia areata; Psoriasis.

Case synopsis

A 24-year-old female with a 2-year history of fistulizing Crohn's disease (CD) was started on adalimumab monotherapy (40 mg biweekly) after failing to respond to various therapy regimens, including mesalazine, azathioprine and systemic corticosteroids. Seven months after treatment initiation she developed erythematosquamous patches on the scalp that evolved with exudative discharge and progressive alopecia (Fig. 1). Subsequently several erythematosquamous patches developed on the lower limbs, palms and soles. She denied personal or family history of psoriasis or alopecia. A skin biopsy obtained from the scalp revealed psoriasiform epidermal changes in association with alopecia areata-like changes in the dermis (Figs. 2-4). Periodic Acid-Schiff stain was negative for fungi. As CD was inactive, adalimumab was discontinued and daily treatment was started with topical clobetasol solution and coal tar shampoo. The scalp lesions cleared with complete hair regrowth within 2 months (Fig. 5). She is currently cleared of scalp lesions for 12 months, but presents occasional flares of scaly plaques on the plantar and internal malleolar regions.

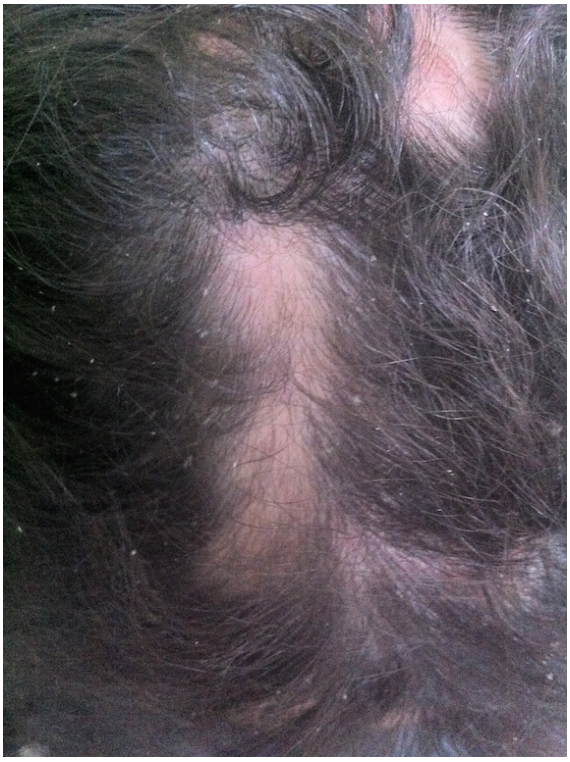
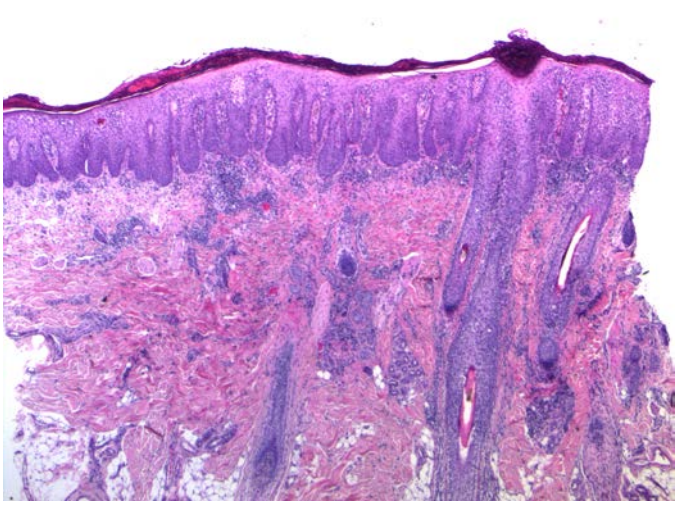


Figure 1. Alopecia patches associated with scalp erythema and psoriasiform scaling during adalimumab treatment.



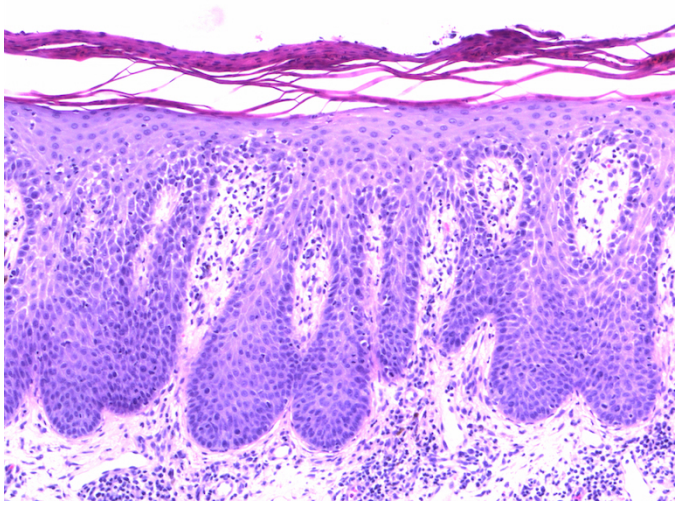


Figure 2. Scalp biopsy showing marked psoriasiform epidermal hyperplasia associated with alopecia areata-like changes in the dermis (H&E, 25x). **Figure 3.** Higher power view of the epidermis highlighting the epidermal hyperplasia with elongation of the dermal papillae, hypogranulosis, and confluent parakeratosis containing neutrophils (H&E, 100x).

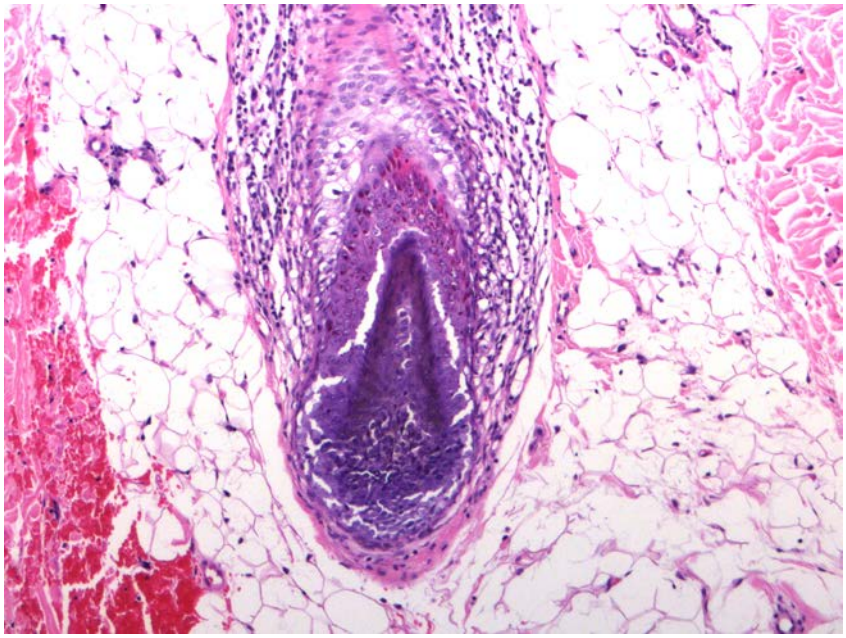


Figure 4. Higher power view illustrating the peribulbar lymphocytic inflammation (H&E, 100x). **Figure 5.** Complete hair regrowth after withdrawal of adalimumab.

Discussion

Psoriasiform eruptions during anti-TNF-α therapy have an estimated prevalence of 1.5 to 5% [1] and usually present as chronic plaque or guttate psoriasis or palmoplantar pustulosis [2]. Although the pathophysiology of this paradoxical effect is not fully understood, two mechanisms have been proposed. In the first, TNF-α blockade increases interferon-α production by plasmacytoid dendritic cells, resulting in activation and amplification of pathogenic T-cells [1,2]. In the second mechanism, TNF-α blockade induces a Th17 immune response and down-regulation of Treg cells [3]. When affecting the scalp, new-onset psoriasis induced by anti-TNF-α agents can result in nonscarring and scarring alopecia [1,2,4]. Histological findings of psoriatic alopecia include psoriasiform epidermal features, along with perifollicular inflammation and atrophy or loss of the sebaceous glands, and, in late stages, destruction of the hair follicle with perifollicular fibrosis [4].

Alopecia is a less well-known side effect of TNF-α inhibitors that has become more recognized in recent years [1,5]. The causes of alopecia occurring during anti-TNF therapy include alopecia areata, psoriatic alopecia, lichen planopilaris, drug-induced lupus erythematosus, androgenetic alopecia, and telogen effluvium [1]. Recently, Doyle *et al* have described a distinct entity that combines clinical and histopathological features of both alopecia areata and psoriatic alopecia, referred to as "psoriatic alopecia/alopecia areata-like reaction secondary to anti-TNF treatment" [5]. Histologically, this form of alopecia is characterized by the presence of psoriasiform epidermal features (acanthosis, hypogranulosis, confluent parakeratosis, and intracorneal neutrophils) and alopecia areata-like dermal changes (marked increase in catagen/telogen and miniaturized follicles and peribulbar lymphocytic inflammation), along with numerous eosinophils and plasma cells [5]. It remains unclear whether this in fact represents a distinct clinical entity or rather a subset of the aforementioned diseases [1].

There is no consensus regarding management of alopecia induced by TNF-α inhibitors and many treatment strategies come from anecdotal experience [1]. For the recently described psoriatic alopecia/alopecia areata-like reaction secondary to anti-TNF-α treatment, in which category our patient seems to fall, cessation of treatment may not be necessarily mandatory, as alopecia has showed significant improvement with topical treatment alone [5,6]. For psoriasis induced by anti-TNF-α, in the presence of severe scalp involvement anti-TNF-α suspension is recommended, and phototherapy and systemic therapies including methotrexate, acitretin and cyclosporine A may be required [1,2,7]. In patients with less extensive psoriasis, a switch to another anti-TNF-α may result in improvement [7]. In both cases, topical therapies (corticosteroids, vitamin D) can be effective [1,7]. In patients who have limited psoriasiform lesions and prefer not to discontinue or switch to another biologic agent, addition of topical therapies, phototherapy or methotrexate may be effective and avoid a change in biologic therapy [7].

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