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Tumor necrosis factor-induced alopecia: alternative pathology and therapy

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Abstract

Tumor necrosis factor (TNF) inhibitors are used to treat Crohn disease and psoriasis. Although they are typically well tolerated, adverse effects include the development of alopecia, and paradoxically, psoriatic lesions. We recently described a woman with Crohn disease who developed alopecia and scalp psoriasis during infliximab therapy. After discontinuing infliximab and beginning oral and topical therapies, her alopecia completely resolved. We compared our experience with that of the Craddock et al. who described a woman with Crohn disease and alopecia secondary to adalimumab therapy. Although the authors described typical histopathologic features of TNF inhibitor-induced alopecia, including decreased sebaceous glands, psoriasiform changes, superficial and deep perifollicular infiltrate of peribulbar lymphocytes, prominent plasma cells, and variable eosinophils, we observed atypical findings that included chronic folliculitis and perifolliculitis with dermal scarring and naked hair shafts in the dermis – reminiscent of folliculitis decalvans. Both patients experienced a complete recovery; however, Craddock et al. described continuing adalimumab therapy and using intralesional triamcinolone acetonide whereas our patient discontinued infliximab therapy, used a combination of topical scalp therapies including betamethasone lotion and mineral oil overnight under occlusion, and began oral minocycline. In conclusion, various histopathologies are observed with TNF-inhibitor induced alopecia and multiple, effective, therapeutic avenues exist for this affliction.

Keywords: alopecia, Crohn, infliximab, psoriasis, scalp, tumor necrosis factor alpha

Introduction

To the Editor: We commend the excellent report by Craddock et al. that describes a 21-year-old woman with Crohn disease who developed tumor necrosis factor (TNF) inhibitor-induced alopecia secondary to adalimumab therapy [1]. We recently published a similar report [2] and have compared our experience with that of the authors.

Our patient was a 23-year-old woman who received infliximab, a TNF inhibitor, to treat her Crohn disease. After 8 months of therapy, she developed an erythematous plaque with diffuse alopecia and overlying scale (**Figure 1**), [2]. The clinical presentation,



Figure 1. The scalp of a 23-year-old woman with Crohn disease who was being treated with infliximab and subsequently developed an erythematous plaque with alopecia and overlying scale.



Figure 2. *The patient had a scalp with normal appearance and resolved alopecia at her 4-month follow-up visit. The hair at the top of scalp is slightly shorter than the surrounding hair due to the new post-alopecic growth.*

as well as our patient's demographics, were very similar to that described by the authors. However, in contrast to Craddock et al.'s patient, whose pathology displayed psoriasiform and alopecia areata-like changes, our patient's scalp biopsy revealed chronic folliculitis and perifolliculitis with dermal scarring and naked hair shafts in the dermis. These changes were consistent with a diagnosis of folliculitis decalvans — a scarring alopecia that typically presents with pustules, inflammatory papules, tunneling hairs, and permanent hair loss [3]. Hence, the pathology of our patient's alopecia did not share the typical features of TNF inhibitor-induced alopecia as were observed by Craddock et al., such as decreased sebaceous glands, psoriasiform changes, and a superficial and deep perifollicular infiltrate of peribulbar lymphocytes, prominent plasma cells, and variable eosinophils [1, 2, 4].

Fortunately, both our patient and that of Craddock et al. experienced a complete resolution of their alopecia after treatment. After developing alopecia, our patient discontinued infliximab therapy, and experienced a full recovery. Ustekinumab was later initiated to control her Crohn's disease and she has not experience a recurrence of her alopecia. Craddock et al. did not discontinue adalimumab and treated their patient with intralesional triamcinolone acetonide 5 mg/mL every month for 3 months [1]. Our patient was successfully treated with oral minocycline and topical 0.05% betamethasone lotion twice daily. In addition,

mineral oil was applied to the scalp overnight under occlusion (with a shower cap); each morning, either 5% coal tar, 2% ketoconazole, or 6% salicylic acid shampoo was used to wash her scalp. Her alopecia completely resolved during the following 4 months (**Figure 2**), [2]. Although psoriasiform eruptions of the scalp may be secondarily infected with pathogenic staphylococcal organisms, our patient's bacterial cultures grew only *Acinetobacter* species that persisted despite minocycline treatment. These were later determined to be commensal bacteria.

In conclusion, whereas our patients are similar, their scalp pathology findings and treatment differed. Yet, both patients experienced a complete resolution of their alopecia. We chose to share our experience in order to demonstrate that the pathology of our patient's scalp alopecia was different than that characteristically observed in patients with TNF inhibitor-induced alopecia. We also suggest an alternative therapy—other than intralesional triamcinolone acetonide—for treating this condition.

References

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