Case Report

Alopecia Areata and Scarring Alopecia Presenting During Golimumab Therapy for Ankylosing Spondylitis

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Golimumab is a human monoclonal antibody useful in the treatment of a wide variety of inflammatory conditions including ankylosing spondylitis, rheumatoid arthritis, psoriatic arthritis, and ulcerative colitis. Golimumab's clinical efficacy is attributed to its inhibition of tumor necrosis factor alpha (TNF-α). As more individuals have been treated with anti-TNF-α therapy, increased numbers of side effects have been identified. We report a patient with a long history of ankylosing spondylitis and atopic dermatitis who noted exacerbation of his alopecia areata early on, and subsequently developed scarring alopecia and facial rash. Clinicians should be aware of the autoimmune reactions associated with golimumab to better inform decisions about therapy and management of underlying rheumatologic disease.
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Key Words: *Golimumab, tumor necrosis factor alpha, lupus, alopecia, drug reaction*

INTRODUCTION

Golimumab has been shown to effectively control many potentially debilitating inflammatory conditions.[1] Cutaneous reactions to anti TNF-α therapy include injection site reactions, nasopharyngitis, infusion reactions reminiscent of serum sickness, infection, heart failure, demyelinating disease, subacute cutaneous lupus erythematosus, and induction of autoimmunity.[2,3] The induction of autoimmunity can be particularly problematic because reactions may mimic the underlying rheumatologic disease being treated. Psoriasiform dermatitis beginning after initiation of therapy is most common, but vasculitis and a lupus-like syndrome can occur. Elevated ANA as well as anti-double stranded DNA antibodies may be encountered in these situations. Experience with other anti TNF-α agents such as adalimumab, etanercept and infliximab has identified differences between reactions to anti TNF-α agents from classical drug-induced lupus erythematosus.[4,5]

REPORT

A 53-year-old man with a long history of ankylosing spondylitis, atopic dermatitis, and localized alopecia areata presented for evaluation for worsening hair loss. His ankylosing spondylitis was initially well controlled with sulfasalazine and later methotrexate. He developed occasional small 1-2 cm areas of alopecia areata over the years, but flares were short lived and limited. He was placed on adalimumab as his disease progressed but then developed treatment resistance at which time golimumab therapy was initiated. He soon noted a flare of alopecia areata that involved approximately 20% of his scalp. Topical minoxidil 2% solution was initiated. He noticed increased photosensitivity accompanied by facial erythema and scaling 3 months prior to presentation (**Figure 1**). Topical desonide as well as tacrolimus cream was of some benefit in controlling the skin changes.

Physical examination revealed two 5 x 6 cm, well-demarcated plaques with obliteration of follicular ostia and fine scale (**Figure 2**). The loss of follicular openings and shiny skin surface indicated the presence of scarring and was different from his prior lesions of alopecia areata which lacked erythema, textural changes of the skin, and loss of follicular openings. The area of involvement with scarring was also in a different location than the lesions of alopecia areata that had favored an ophiasis distribution. He had scaling and erythema of the face in a photodistribution that was typical of acute cutaneous involvement with lupus. His skin examination was otherwise unremarkable.

The patient elected to continue golimumab treatment because of the good control of his ankylosing spondylitis, and manage his alopecia and skin findings with local treatment. Sun protection, clobetasol solution applied to the scalp once or twice daily as needed, and monthly intralesional triamcinolone acetonide injections (10 mg/cc), 0.8 cc to the two plaques led to significant improvement. Serological studies failed to identify anti-histone antibodies but revealed anti double stranded DNA antibodies. The patient continues on this regimen and is satisfied with his progress.

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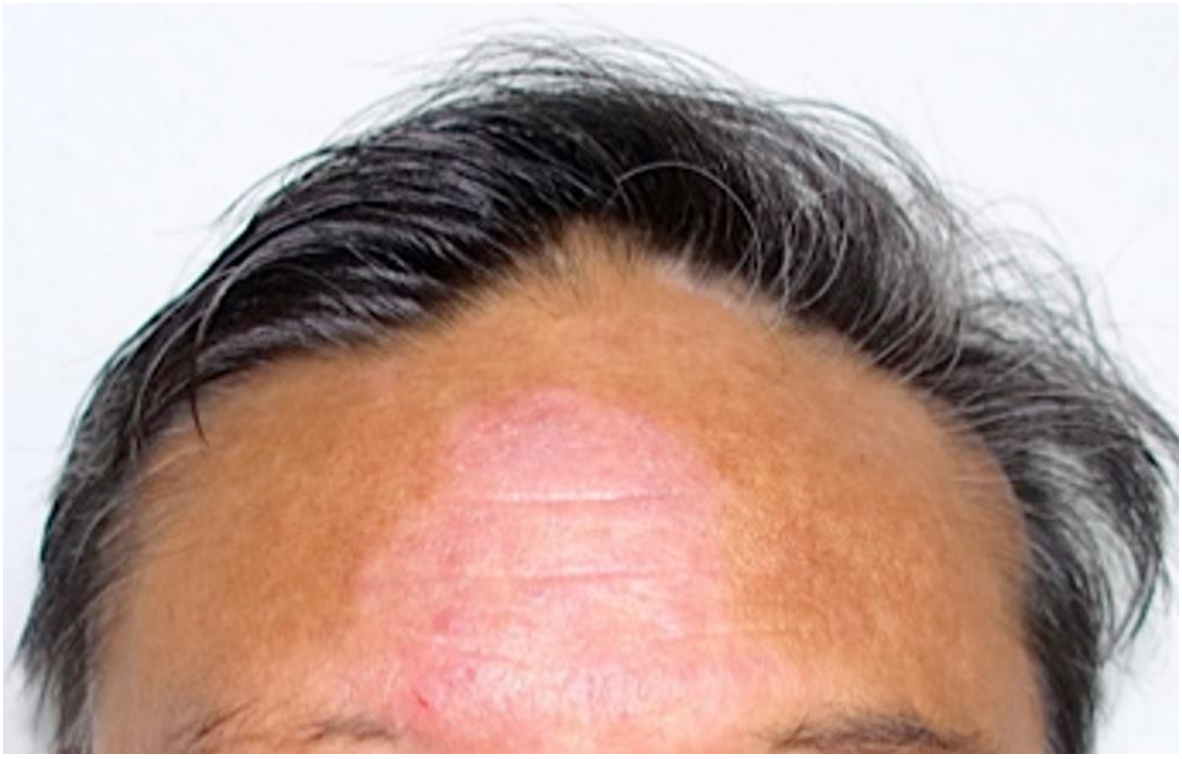

Figure 1. Erythema and scaling are noted on the forehead.

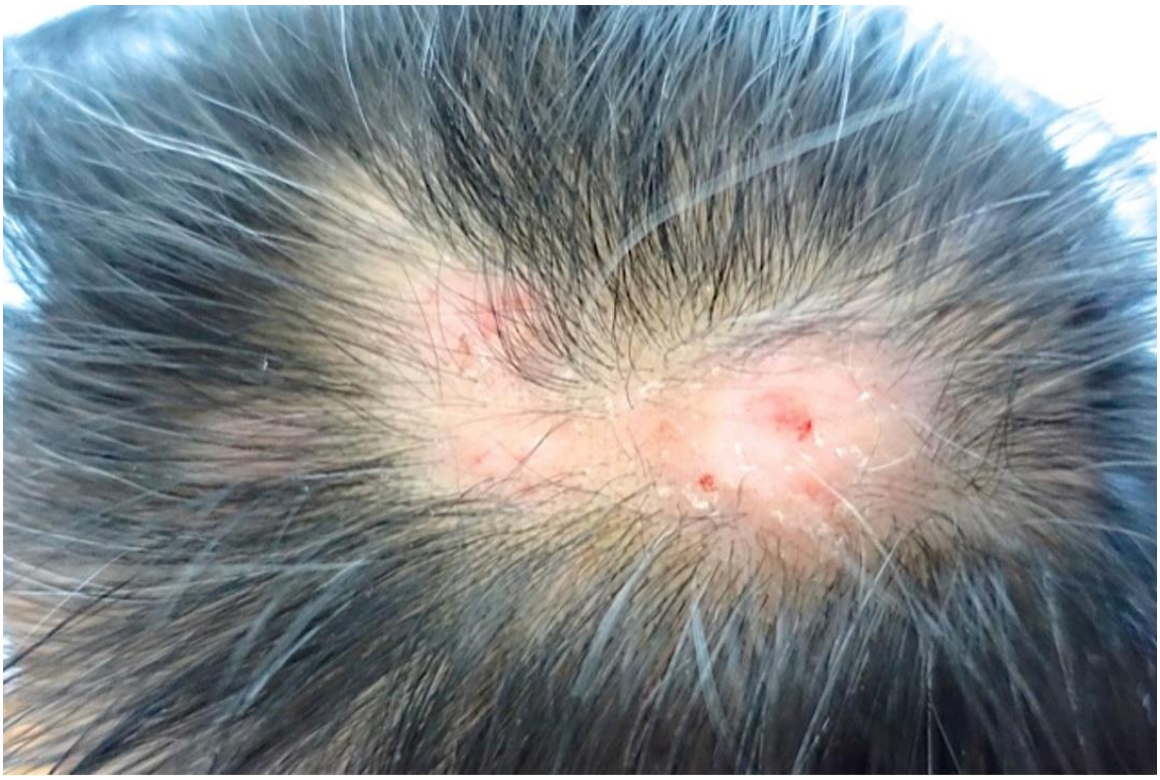

Figure 2. Examination of the scalp reveals a scarred plaque with loss of follicular openings.

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DISCUSSION

Anti-TNF-α therapy has been found to be very useful for rheumatic disease but has been associated with a variety of side effects that include drug-induced lupus erythematosus-like changes. Classic drug-induced lupus typically affects older individuals, has an approximately equal ratio of affected males and females, is associated with anti-histone antibodies, and fails to demonstrate hypocomplementemia. The overall risk of drug-induced lupus in the setting of anti TNF-α therapy is considered very low and demonstrates some important differences from classic drug-induced lupus erythematosus.[5] Drug-induced lupus in the setting of anti TNF-α therapy affects females more often than males, is associated with antihistone antibodies and hypocomplementemia with a frequency similar to idiopathic systemic lupus erythematosus.[5] This can create a confusing clinical picture for individuals unfamiliar with drug-induced lupus arising in the setting of anti TNF-α therapy. We believe that our patient's flares in alopecia areata can be attributed to immune dysregulation and that the scarring lesion which developed later were different and represented a manifestation of cutaneous lupus erythematosus as did his facial eruption which was limited to a photodistribution. The clinical findings in drug-induced lupus arising in the setting of anti TNF-α therapy can exactly mimic the cutaneous changes of idiopathic lupus. Correlation with the timing of medication is important in establishing the diagnosis. Cases in the literature of lichen planopilaris or psoriasiform dermatitis associated with alopecia arising in the setting of anti TNF-α therapy may represent a spectrum of findings similar to the changes in our patient.[6,7] Both subacute cutaneous lupus erythematosus and psoriasiform lesions are now well recognized in the setting of anti TNF-α therapy but the mechanisms of these changes are still not completely understood.[8]

Because of the effective control of serious underlying debilitating disease, individuals may opt to stay on therapy. Early recognition of cutaneous changes can allow for prompt institution of therapy in order to mitigate morbidity. Clinicians also should be alert to other kinds of changes that have been noted such as uveitis, interstitial drug reaction, and inflammatory myopathy. A comprehensive approach to identifying side effects not only helps individuals on therapy but furthers our understanding of anti-TNF-α actions.

CONFLICT OF INTEREST DISCLOSURE

The authors have no conflicts of interest to disclose.

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