Treatment of lichen planopilaris with adalimumab in a patient with hidradenitis suppurativa and rheumatoid arthritis

Maryam Shayesteh Alam, MD, FRCPC, FAAD, and Britannie LaBelle, MD

Barrie, Ontario, Canada

Key words: adalimumab; frontal fibrosing alopecia; hair regrowth; hidradenitis suppurativa; lichen planopilaris; rheumatoid arthritis.

INTRODUCTION

Adalimumab (Humira; Abbott Laboratories, Puerto Rico) is a recombinant human IgG1 monoclonal antibody for tumor necrosis factor and is a treatment option for both hidradenitis suppurativa (HS) and rheumatoid arthritis (RA). We report a case of a patient with lichen planopilaris (LPP) that had hair regrowth when treated with adalimumab, originally prescribed to the patient for RA and HS.

CASE STUDY

A 61-year-old woman was referred with a 2-year history of painful nodules and abscesses located on her groin, along with a 4-month history of patchy hair loss on her scalp. The patient’s medical history included RA and Sjögren syndrome.

The patient was under the care of a rheumatologist for RA diagnosed in 2011. Her RA was first managed with methotrexate. However, with the increase in her liver enzymes, methotrexate was stopped in 2011 and replaced with hydroxychloroquine (Plaquenil; Sanofi-Synthelabo Inc, Paris, France), 200 mg twice a day Monday to Friday. Leflunomide, 20 mg once a day, was added to her treatment plan in 2012, and sulfasalazine, 1000 mg twice a day in 2014. In 2015, hydroxychloroquine was replaced with certolizumab pegol (Cimzia; UCB Inc, Smyrna, GA), 400 mg every 2 weeks. When the patient was referred to our clinic, she was on triple therapy of certolizumab pegol, leflunomide, and sulfasalazine for her RA.

Examination of the patient’s groin presented characteristics of Hurley stage 2 HS, including comedones, active inflammatory nodules, and abscesses with drainage and scarring. The patient received a treatment of clindamycin, 300 mg twice a day, and rifampin, 600 mg once a day for 30 days for her HS.

The patient’s scalp had patches of scarring alopecia in the frontal hairline, a receding hairline, and follicular hyperkeratosis consistent with frontal fibrosing alopecia (Fig 1, A). The frontal and parietal areas of the patient’s scalp presented nonpruritic, multifocal patches of early scarring alopecia with red scaly patches suggestive of LPP (Fig 2, A).

Scalp biopsies found scarring in the dermis with loss of hair follicles with perifollicular inflammatory infiltrate of lymphocytes and some perifollicular fibrosis consistent with LPP. The Periodic acid–Schiff stain did not show thickening of the basement membrane and there was no vacuolar change to suggest discoid lupus erythematosus. The patient was prescribed hydroxychloroquine, 200 mg twice a day Monday through Friday, and clobetasol 0.05% scalp lotion twice a day for 3 months for her LPP.

At 2-month follow-up examination, the patient’s LPP patches were not improved, and HS was resistant to clindamycin and rifampin. Therefore, certolizumab pegol and sulfasalazine were both discontinued, and the patient was started on adalimumab (160 mg week 1; 80 mg week 2; 40 mg week 3; 40 mg week 4; 20 mg week 5).
weekly beginning at week 4). She continued to receive leflunomide therapy.

During the next follow-up visit, the patient had been on adalimumab for 3 months and reported improvement in both HS and RA. On examination of the groin, hypertrophic scars were present, and there were no active HS nodules or abscesses. On examination of the scalp, LPP patches had hair regrowth and a reduction in redness. Further improvement of hair regrowth has been noted in the latest follow-up examination 6 months later (Figs 1, B and 2, B).

**DISCUSSION**

Therapeutic management of LPP is challenging with current therapeutic options failing to alleviate active inflammation and prevent disease progression.1-3 Most remarkably, improvement of
hair regrowth and reduction in inflammation of LPP patches were observed in this case. Improvement to hair regrowth comparable to that in this case has not been reported in the literature when patients were treated exclusively with hydroxychloroquine or with a combination of hydroxychloroquine and topical corticosteroids. Furthermore, studies have found that no patient had visible hair regrowth on hydroxychloroquine, and in some cases, patients continued to have progressive hair loss. Therefore, it is unlikely that the hair regrowth seen in this patient can be attributed to hydroxychloroquine alone, and it is for this reason that we suggest that adalimumab is responsible. The possibility of a combined therapeutic effect of adalimumab and hydroxychloroquine attributing to the physiologic findings may also be worth exploration.

Adalimumab has already been reported to yield positive results in the management of cutaneous and oral lichen planus. Additionally, in a case report adalimumab was shown to successfully treat therapy-resistant LPP and folliculitis decalvans; however, hair regrowth was not reported or observed in photos.

The cause and pathogenesis of LPP are unknown, and recommended topical and systemic treatments often give unsatisfactory results. This case report highlights the promise for further understanding of this condition and its treatments. We suggest further investigation of adalimumab and hydroxychloroquine for the treatment of LPP.

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