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Treatment of Dissecting cellulitis of the Scalp with Tildrakizumab

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Keywords: Dissecting cellulitis of scalp, cicatricial alopecia, follicular occlusion, Tildrakizumab

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Introduction

Dissecting cellulitis of the scalp (DCS) is an inflammatory disease that classically presents with multiple tender fluctuant nodules with interconnecting sinuses on the vertex or occipital scalp. Initially there is an overlying non-scarring alopecia. Treatment delay leads to cicatricial alopecia (1). DCS is associated with acne conglobata (AC), hidradenitis suppurativa (HS) and pilonidal sinus. Collectively the conditions form the follicular occlusion tetrad. We present a case of DCS successfully treated with Tildrakizumab, an anti-interlukin-23 (IL-23) monoclonal antibody; implicating a role for the T-Helper 17 (TH17) immune axis in the pathogenesis of DCS.

Case Report

A 28-year-old man with a past-history of HS and AC presented with several large, fluctuant, tender nodules on the scalp with overlying alopecia (Figure 1A). Initial treatment with Isotretinoin 20mg, erythromycin 500mg and intralesional triamcinolone injections was unsuccessful. Two doses of subcutaneous injection of Tildrakizumab 4 weeks apart produced a significant reduction in the number and severity of pustules and alleviated scalp tenderness along with hair regrowth in the areas of alopecia (Figure 1B).

Discussion

DCS is frequently refractory to commonly used agents including broad spectrum antibiotics, isotretinoin, dapsone and zinc sulphate. DCS shares a similar pathogenesis to HS and has been associated with an increased secretion of Tumour necrosis Alpha (TNF-α) and Interlukin-6 (IL-6). There have been several case reports of successful treatment of DCS with TNF-α inhibitors infliximab and adalimumab, agents which have been used to treat HS(2). In addition, Secukinumab, an interlukin-17 (IL-17) inhibitor was successfully used to treat a patient with recalcitrant DCS (3).

The pathophysiology of the follicular occlusion tetrad involves follicular keratinization and subsequent occlusion with acute neutrophilic and chronic granulomatous inflammation of hair follicles. Consequential follicular rupture and abscess formation ultimately leads to annihilation of the entire pilosebaceous unit(4).

Other factors implicated in the pathogenesis of HS that might be relevant to DSC include altered toll like receptors in dendritic cells and macrophages leading to increased secretion of IL-17 and TNF that in turn increase secretion of IL-23(5, 6). Successful treatment of DCS with Tildrakizumab may implicate IL-23 and the TH17 axis in the pathogenesis of this potentially debilitating condition.

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Figure 1. **A**, Appearance of hair before treatment with Tildrakizumab demonstrating scalp cysts and decreased hair density. **B**, Appearance of hair following 8 weeks of Tildrakizumab therapy showing increased hair density and reduced number of pustules

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